MAR 31 1945

Medical Library

VOLUME 69 NUMBER 2

AMERICAN JOURNAL

OF

DISEASES OF CHILDREN

EDITORIAL BOARD

CLIFFORD G. GRULEE, Chief Editor
Box 672, Evanston, Ill.

OSCAR M. SCHLOSS, New York
FRITZ B. TALBOT, Boston
A. A. WERCH, Cincinnati
H. F. HELMHOLZ, Rochester, Minn.
FRANCIS SCOTT SMYTH, San Francisco
JAMES L. WILSON, Ann Arbor, Mich.

FEBRUARY 1945

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO 10, ILLINOIS. ANNUAL SUBSCRIPTION, $8.00

Entered as Second Class Matter Jan. 4, 1911, at the Postoffice at Chicago, Under the Act of Congress of March 3, 1879

COPYRIGHT, 1945, BY THE AMERICAN MEDICAL ASSOCIATION

CONTENTS OF PREVIOUS NUMBER

JANUARY 1943. NUMBER 1

Random Thoughts on the Position of Pediatrics: Presidential Address. James L. Gamble, M.D., Boston.

Alum-Precipitated Diphtheria Toxoid for Immunization of Persons Exposed to Whooping Cough. Jesse Muñoz Turnbull, M.D., Mérida, Mexico.

Psychic Trauma of Operations in Children and a Note on Combat Neurosis. David M. Levy, M.D., New York.

Resistance of Cotton Rats to the Virus of Poliomyelitis as Affected by Intake of Vitamin B Complex, Partial Inanition and Sex. H. M. Weaver, with the Technical Assistance of Norma Hastings and Helen Ammon, Detroit.

Case Reports:
Generalized Vaccinia in an Eczematous Child: Demonstration of Virus and Comment on "Kaposi's Varicelliform Eruption." Rufus E. Hershey, M.D., and William E. Smith, M.D., Denver.

Progress in Pediatrics:
Salicylate Poisoning: Report of Four Cases. Mary Mahar Trail, M.D., and Maud L. Menten, M.D., Ph.D., Pittsburgh.

News and Comment.

Abstracts from Current Literature.

Society Transactions:
Chicago Pediatric Society and Children's Division of Cook County Hospital.

Book Reviews.

Directory of Pediatric Societies.

American Journal of Diseases of Children

LUME 69 FEBRUARY 1945 NUMBER 2

COPYRIGHT, 1945, BY THE AMERICAN MEDICAL ASSOCIATION

CLINICAL APPRAISAL OF INFANTS' HEAD SIZE

JULIAN D. BOYD, M.D.

IOWA CITY

In comparison with the body as a whole the ntral nervous system grows precociously dur- prenatal life and the period of infancy.[1] bout 75 per cent of the bulk of the brain has en established by the conclusion of the first ar of life. Injury to the developing brain may nifest itself functionally or structurally. In me instances injury to the brain incurred at rth or during infancy does not make itself ob- ous until malfunction becomes apparent at a er age. In other instances, however, injury the brain affects the rate of growth of the anium. It follows, then, that if the physician s a means of appraising accurately the degree d rate of growth of the head of an infant, he en will be able to discern damage to the central rvous system earlier than otherwise would be ssible.

Measurement of the circumference of the head considered a part of the routine physical amination of infants. Pediatric textbooks fer tables of average head size in terms of age d of sex but provide little information valuable determining the significance of deviations from ese average values. To make possible better sight into the rate and pattern of growth of fants' heads, the serial records of a group of resumably normal babies observed in the de- rtment of pediatrics of the College of Medicine the State University of Iowa have been re- ewed.

SUBJECTS AND METHODS

The subjects employed were chosen from those udied in the metabolism ward of the department pediatrics. All infants whose records included easurements of head circumference as well as height d weight were included. The group comprised 100 fants, of whom 81 were boys and 19 girls. With a ngle exception, multiple serial measurements were ailable for each subject. In all, approximately 650 ts of individual measurements were included, each pressing body weight, body length and head circumference. The median number of serial measurements was six, and the median interval between measurements was four weeks. Measurements were made by graduate nurses who had been trained in anthropometric technics, and standard equipment for measurement was employed. Head circumference was determined through the use of a steel tape measure passed over the greatest prominence of the occiput and of the glabella. All measurements were made and recorded in metric units.

The infants studied were recruited mostly from orphanages or from the maternity ward of the obstetric service of the State University of Iowa. The majority were first-born children, and most were children of unmarried mothers. A significant number, however, were children of young university student couples, placed in the hospital because the mothers desired to continue employment outside the home. When possible the infants were placed under the ward regimen of care and of diet soon after birth; most entered the ward when they were not more than 4 months old, and the majority were not older than a month when first observed. All subjects were of the white native stock typical of the midwestern population. Each infant received excellent personalized care and attention from graduate nurses and from other attendants especially trained in the care of normal babies. Various regimens of diet were employed during the eleven years encompassed by this study. Each of these regimens was designed to meet good standards of infant nutrition except for an experimental variation in the amount of vitamin D. In some groups the intake of vitamin D proved to be suboptimal; the growth of these infants in length was retarded as compared with that of other groups. In general, the type of nutritional regimen was constant for each subject throughout his period of observation. For the most part the infants were healthy and active, living in a manner not essentially different from that of an infant in a good home. In numerous instances infections of the upper part of the respiratory tract and of the ear developed, however, and some of the subjects were ill for notable periods. Some babies eventually were discarded as subjects of metabolic study because of their recurrent or persistent infections or because of their inability to ingest regularly the diet offered them.

PRESENTATION OF DATA

Table 1 gives the mean head circumference for boys and girls at progressive ages from 1 to 12 months, together with the standard deviation, the standard error of the mean and the average rate of monthly increment. Table 2 offers mean head size in terms of body length instead of age. It will be noted that in each table the data follow

From the Department of Pediatrics, College of edicine, State University of Iowa.

Presented at the Fifty-Fifth Annual Meeting of e American Pediatric Society, Sept. 27, 1944, Atlantic ity, N. J.

1. Hesdorffer, M. B., and Scammon, R. E.: Proc. oc. Exper. Biol. & Med. **33**:414 (Dec.) 1935.

an orderly trend but that there is less deviation within each segment when head size is related to body length than when it is related to age. There is a notable sex difference in head size in terms of age, but this disappears when related to body length. The successive increments for head size follow different patterns in the age analysis from those of the length analysis. The increment rate tends to decline in later infancy in much

The striking interdependence of the data led expression of the trend by means of empirical derived formulas. The two dotted lines supe imposed on charts 1 and 2 and on other char to be cited subsequently represent respectiv the formulas Hd = 0.4L + 15 and Hd = 0.5L 10, in which Hd refers to head circumference a L to body length, each expressed in centimete The lines representing graphically the two fo

TABLE 1.—*Head Circumference of Infants in Relation to the Mean Values for the Average Age in Months*

Boys						Girls						Difference of Means for Boys and Girls
Average Age, Mo.	No. of Observations	Head Circumference, Cm.			Increment, Cm. per Mo.	Average Age, Mo.	No. of Observations	Head Circumference, Cm.			Increment, Cm. per Mo.	
		Mean	S.D.	S.Em.				Mean	S.D.	S.Em.		
1	55	36.0	1.4	0.20		1	16	35.0	0.81	0.21		1.0
2	65	38.9	1.45	0.17	2.9	2	17	37.2	1.26	0.32	2.2	1.7
3	71	39.5	1.4	0.17	0.6	3	16	38.5	1.17	0.30	1.3	1.0
4	65	40.6	1.5	0.19	1.1	4	16	39.4	1.07	0.28	0.9	1.2
5	61	41.6	1.2	0.15	1.0	5	11	40.3	0.93	0.29	0.9	1.3
6	64	42.4	1.15	0.15	0.8	6	13	41.2	0.77	0.22	0.9	1.2
7	42	42.9	1.2	0.19	0.5	7	7	42.3			1.1	0.6
8	40	43.7	1.1	0.17	0.8	8	6	42.8			0.5	0.9
9	24	44.3	1.1	0.22	0.6	9	4	42.7			—	1.6
10	27	44.9	1.1	0.22	0.6	10	—					
12	24	45.5	1.2	0.25	0.3	12	4	44.9			0.5	0.6
Average difference of means for boys and girls												1.0 cm.

TABLE 2.—*Head Circumference of Infants in Relation to the Mean Value for Average Body Length in Centimeters*

Average Body Length, Cm.	Boys				Girls				Average Increment for Each Cm. of Length	
	No. of Observations	Head Circumference, Cm.			No. of Observations	Head Circumference, Cm.				
		Mean	S.D.	S.Em.		Mean	S.D.	S.Em.	Boys	Girls
50	..	....	...	....	6	34.6	0.6	0.29		
51	12	34.7	0.9	0.28					....	0.15
52	..	....	...	....	7	34.9	1.2	0.51	0.75	
53	26	36.2	1.2	0.24	..	....	...	....	....	0.30
54	..	....	...	....	7	35.6	1.1	0.48	0.45	
55	25	37.1	0.9	0.19	..	....	...	....	....	0.60
56	..	....	...	....	14	36.8	1.4	0.38	0.40	
57	41	37.9	1.4	0.21	..	....	...	....	....	0.75
58	..	....	...	....	14	38.3	0.8	0.22	0.40	
59	46	38.7	1.2	0.18	..	....	...	....	....	0.40
60	..	....	...	....	11	39.1	1.1	0.35	0.55	
61	44	39.8	1.1	0.16	..	....	...	....	....	0.45
62	..	....	...	....	12	40.0	1.2	0.37	0.45	
63	62	40.7	1.0	0.13	..	....	...	....	....	0.40
64	..	....	...	....	11	40.8	1.1	0.36	0.40	
65	59	41.5	0.9	0.12	..	....	...	....	....	0.40
66	..	....	...	....	14	41.4	1.0	0.28	0.40	
67	52	42.3	1.2	0.17	..	....	...	....	....	0.45
68	..	....	...	....	9	42.3	0.9	0.34	0.35	
69	56	43.0	1.0	0.14	..	....	...	....	....	0.30
70	..	....	...	....	6	42.9	1.2	0.55	0.50	
71	40	44.0	1.1	0.17	..	....	...	....	0.05	
73	28	44.1	0.8	0.16	..	....	...	....	0.40	0.30
75	19	44.9	1.0	0.24	2	44.5	...	....	0.30	
77	13	45.5	0.8	0.24	..	....	...	....		
78.5	11	45.5	0.7	0.22						
Average increment for each centimeter of body length up to 75 cm.									0.42	0.40
Median increment									0.4 cm.	0.4 cm.

greater degree when head size is related to age than when it is related to body length. The relationship between head size and body length is demonstrated clearly by graphic means.

Charts 1 and 2 show the dispersion of observed values for head circumference and for body length, of boys and girls respectively. Each dot represents a single set of measurements for a given infant. Included in the scattergrams are the serial measurements of all of the infants studied.

mulas were designed to intersect at a point cor responding to a head size of 35 cm. and a bod length of 50 cm. These values are generall accepted as representative for a boy born at ter whose weight approximates 3,500 Gm. The zon encompassed by these formulas includes th majority of measurements, particularly after length of 55 cm. has been attained. Because this, it was inferred that these formulas migh serve as a gage of normality of head size for given body length during the period of infanc

To test further the validity of this premise, ta reported by others were reviewed and com- red with the calculated values based on the egoing formulas. Two sets of data seemed apted for such comparison and adequate for e purpose. One of these, relating to Iowa in- nts of similar age, was reported by the Iowa ild Welfare Research Station in 1931.[2] The her data were reported by Vickers and Stuart 1943 and were based on measurements of fants from the vicinity of Boston.[3] Each of ese two studies offers average height and aver-

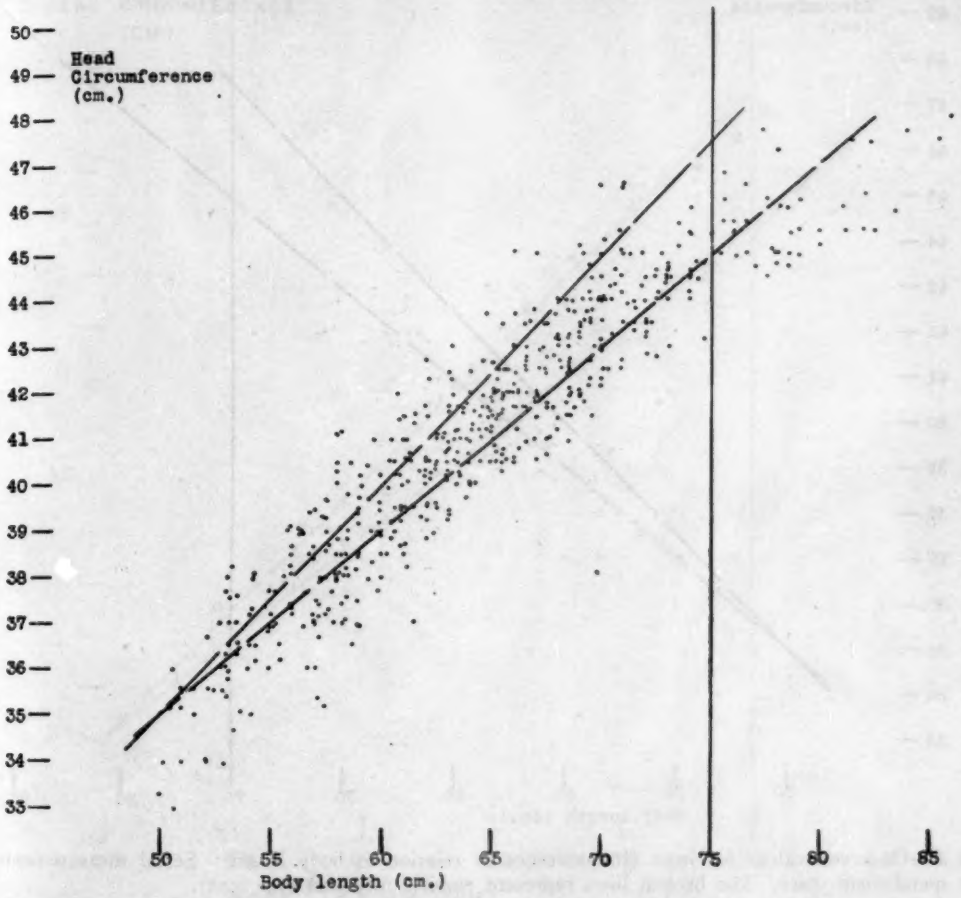


Chart 1.—Observed values for head circumference in relation to body length: Serial measurements of boys rom the metabolism ward. The broken lines represent empiric formulas (see text).

ge head circumference in terms of advancing ge throughout infancy. The study by Vickers nd Stuart offers in addition separate listing of he decile distribution for head size in relation o age and for body length in relation to age. Neither study offers values for head size in rela- ion to body length. However, in view of the trong correlation noted in the present study between head size and body length, it has been assumed tentatively that in the Vickers and Stuart study the low and the high decile groups for body length comprised essentially the same population as was included in the corresponding decile groups for head sizes. The statistical error inherent in such an assumption is recognized, but the comparison does not seem unjustifiable or invalid.

Chart 3 shows the average values for head circumference of boys in relation to their body length as calculated from the data of the Iowa Child Welfare Research Station,[2] of the study of Vickers and Stuart [3] and the present study of subjects from the metabolism ward. Superimposed are the values for head size in relation to body length as calculated from the empiric formulas already described. Note that the data from this study tend to conform rather closely to the formula $Hd = 4L + 15$, whereas the other two sets of data fall more nearly in accordance with the formula $Hd = 0.5L + 10$. The greatest discrepancy noted is in the shorter subjects of the group from the metabolism ward.

2. Physical Traits of Iowa Infants, Iowa Child Welfare Station, State University of Iowa, Am. J. Dis. Child. **42**:1137 (Nov.) 1931.

3. Vickers, V. S., and Stuart, H. C.: J. Pediat. 2:155 (Feb.) 1943.

To check further the correlation between head circumference and body length, the values for the tenth, fiftieth and ninetieth deciles from Vickers and Stuart's data have been matched for head size in relation to body length for boys and for girls in their series and expressed graphically in chart 4. Here the curves tend to approximate the formula $Hd = 0.5L + 10$ and to be essentially superimposed not only for decile distribution but for sex. The formula is applicable up to a body length of 70 cm. in Vickers and Stuart's data and is not violated notably up to a body length of 75 or 80 cm., corresponding roughly to the span of the first year of life. Here again the shorter groups tend to fall away from the calculated head size earlier than do the taller groups; girls, inasmuch as they tend to be shorter for their age than boys, tend to depart from the calculated head size after completion of the first year of life sooner than do boys of average body length. Beyond the body length of 75 cm. the predicted values for all young children tend to

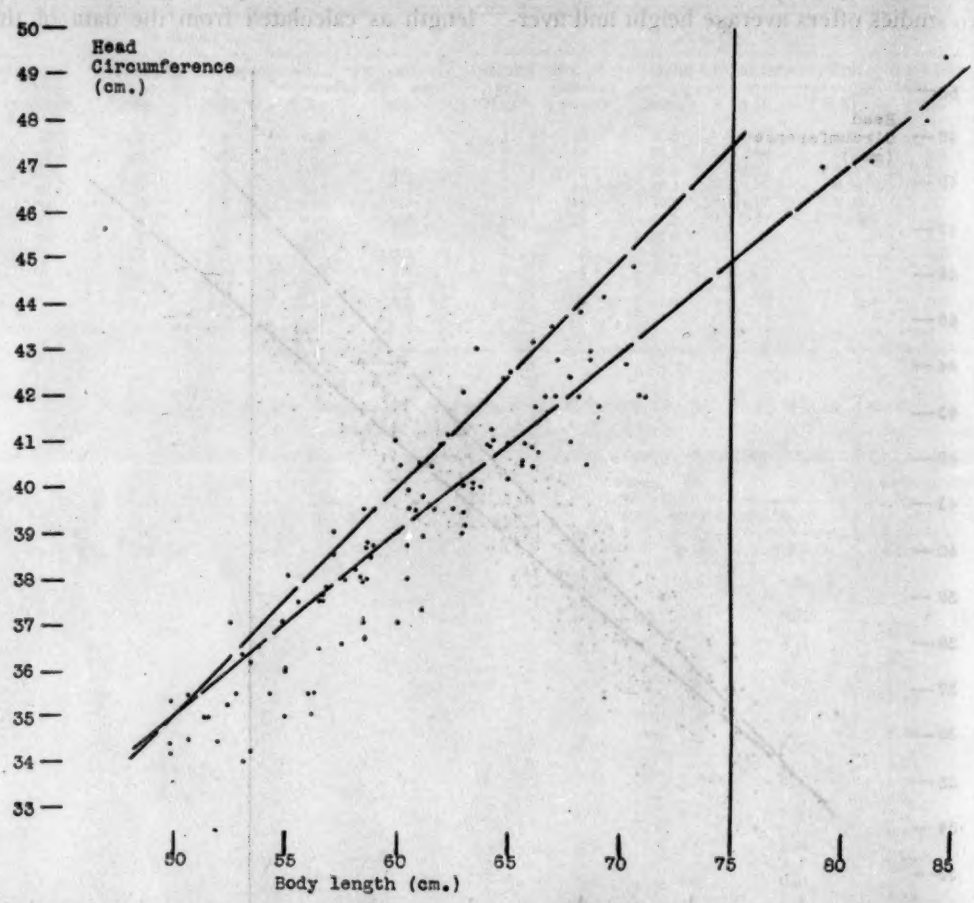


Chart 2.—Observed values for head circumference in relation to body length: Serial measurements of girls from the metabolism ward. The broken lines represent empiric formulas (see text).

TABLE 3.—*Comparison of Average Measurements at Successive Ages for Boys Who Were Immature at Birth and Those Who Presumably Were Born at Full Term*

Average Age, Mo.	Boys Weighing Less * Than 3,100 Gm. at Birth							Remaining Male Infants of Series						
	No. of Observations	Body Length, Cm.			Head Circumference, Cm.			No. of Observations	Body Length, Cm.			Head Circumference, Cm.		
		Mean	S.D.	S.Em.	Mean	S.D.	S.Em.		Mean	S.D.	S.Em.	Mean	S.D.	S.Em.
1	20	51.7	1.7	0.39	34.9	1.3	0.30	32	53.9	1.7	0.31	36.6	1.1	0.20
2	21	55.7	1.7	0.37	37.4	1.3	0.29	47	57.6	1.6	0.24	38.2	1.2	0.18
3	18	58.8	1.7	0.42	38.8	1.4	0.35	44	60.6	2.2	0.33	39.7	1.9	0.29
4	21	60.9	1.8	0.41	39.8	2.2	0.49	52	63.3	2.3	0.32	40.8	1.1	0.16
5	19	63.6	1.8	0.42	40.8	1.2	0.29	39	65.4	1.8	0.30	41.8	1.0	0.16
6	22	65.5	1.9	0.42	41.8	1.2	0.27	39	67.6	2.0	0.33	42.6	0.9	0.15
7	15	67.1	2.2	0.58	42.1	1.4	0.38	29	69.6	2.1	0.40	43.2	1.1	0.20
8	14	68.7	2.4	0.67	43.0	1.1	0.32	29	71.3	2.2	0.42	43.8	0.8	0.16
9	9	69.9	2.4	0.86	43.9	0.2	0.08	18	72.7	2.6	0.63	44.5	0.9	0.22
10	7	71.2	2.6	1.0	44.3	1.2	0.49	18	74.2	3.2	0.77	45.0	0.9	0.23
11	3	73.5	2.0	1.4	45.1	0.9	0.63	11	76.2	2.5	0.80	45.7	1.0	0.32
12														
13	..	....	...	....	....	...	....	18	77.6	...	....	45.9		

* Average weight, 2,800 Gm.

ceed the observed measurements by progressely greater amounts. This is observable also the scattergrams of values for the infants from metabolism ward, as is illustrated in charts and 2.

Next, in an attempt to appraise the effect of tial smallness of stature on the subsequent ttern of head growth, the subjects from the tabolism ward were reviewed as to their size the time of birth. Of the 100 subjects, records weight at birth were available for 65. Of this

sideration for those who weighed less than 3,100 Gm. at birth and those who offered no evidence of immaturity. Included as well are average values for body length. Chart 5 presents the data as to the relation between age and body length for the two sub groups, and chart 7 as to the relation between head circumference and age.

The guide lines in chart 5 represent dimensions of presumably normal boys, compiled from data published by the Iowa Child Welfare Research Station [4] and used routinely in this clinic

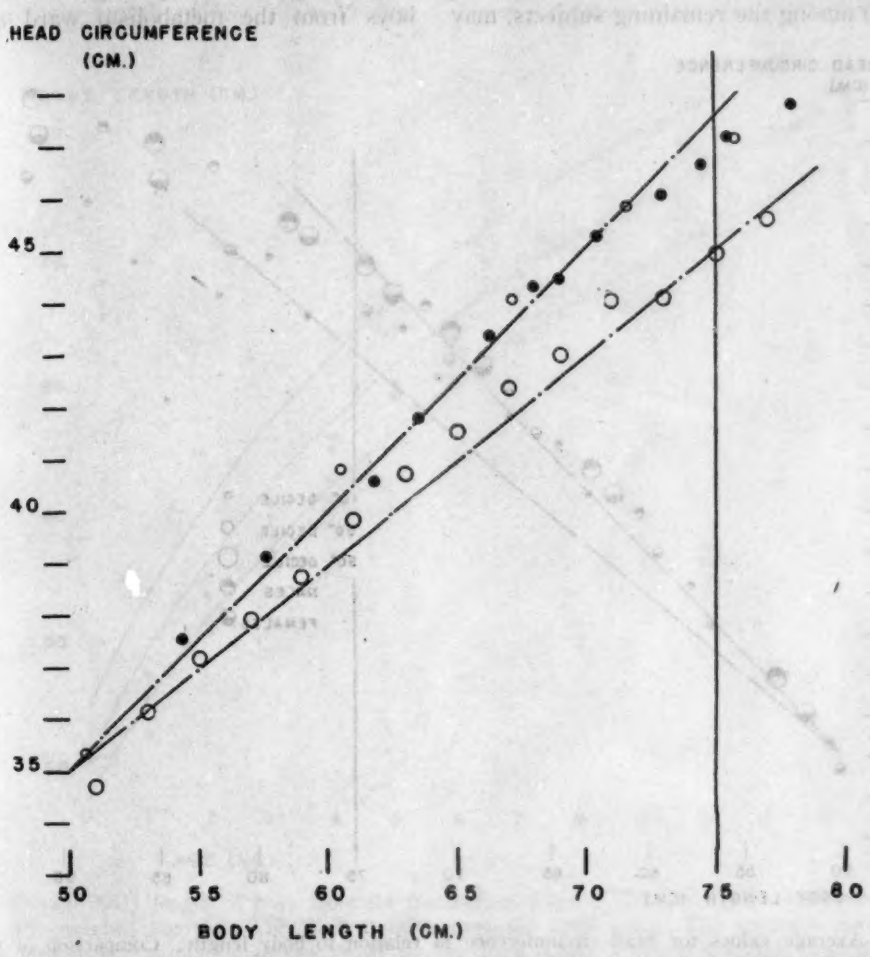


Chart 3.—Average values for head circumference in relation to body length: Data for subjects from the etabolism ward (large white circles) compared with data reported by the Iowa Child Welfare Research Station lack circles) and those reported by Vickers and Stuart (small white circles). The broken lines represent mpiric formulas (see text).

roup 28 had weighed not more than 3,100 Gm., r 6 pounds and 13 ounces, at birth; some had eighed less than 2,500 Gm., and the average eight at birth for the 28 subjects was 2,800 m. In addition, 3 subjects whose weight at birth as not known had been so small at the time of eir earliest recorded measurements that in all robability they too had been immature at the me of birth. In table 3 the data for head size boys have been retabulated with separate con-

for appraisal of infants. The standard curves represent the mean values together with one standard deviation above and one below the mean. Note that the serial values for height of the infants who were immature at birth tend to

4. Meredith, H. V.: The Rhythm of Physical Growth, Studies in Child Welfare, Iowa City, University of Iowa, 1935, vol. 11, no. 3. Boynton, B.: The Physical Growth of Girls, ibid., 1936, vol. 12, no. 4.

approximate the curve representing one standard deviation below the mean. This position is maintained by the initially immature group with fair constancy throughout the limits of the recorded data. The group comprising the infants presumably born at term follows a different trend: Their values tend to fall below the average during the earlier months of infancy, but from the sixth month onward they tend to surpass the average. The initial comparative smallness of the infants presumably born at term, as well as the high incidence of immaturity at the time of birth among the remaining subjects, may

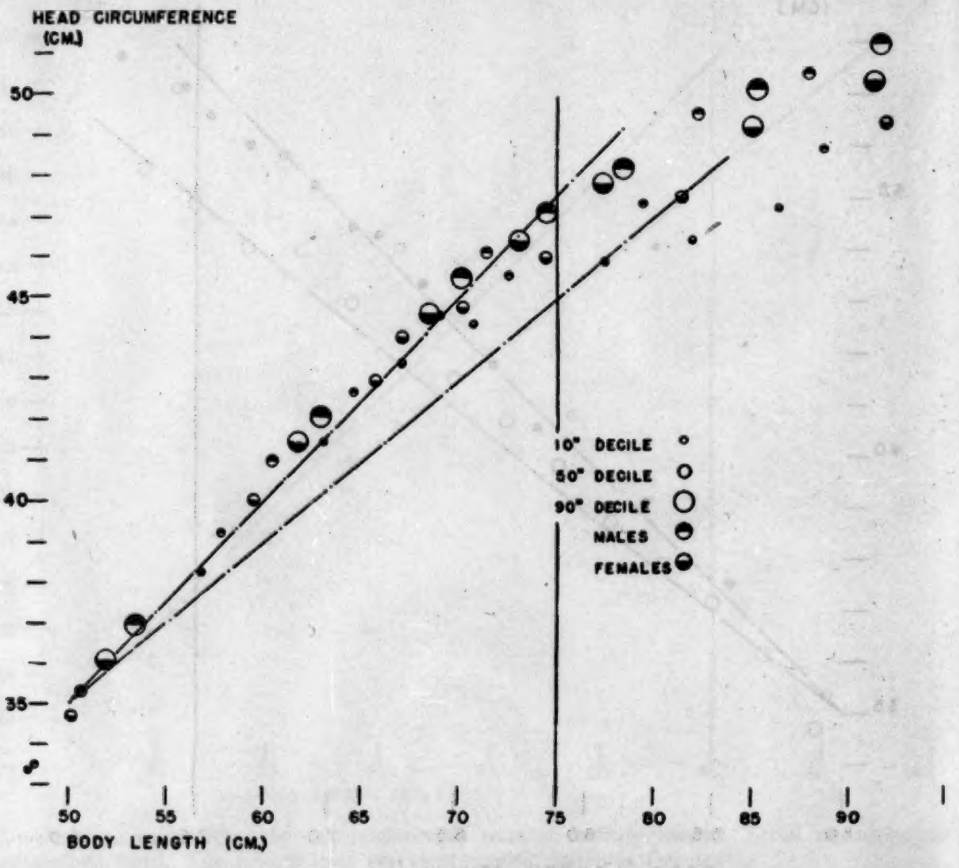


Chart 4.—Average values for head circumference in relation to body length: Comparison of values for the tenth, fiftieth and ninetieth decile groups from Vickers and Stuart's data. The broken lines represent empirical formulas (see text).

be explained at least in part by the nature of the subject personnel: Most of the infants were from first pregnancies, and most of the mothers lived in a substandard socioeconomic status during their pregnancy. Furthermore, those infants who were discarded as subjects of metabolic study after an unsatisfactory period of trial do not contribute to the data for the later months and yet tend to weight downward the averages during the months of inclusion.

As would be expected, the head size of initially small babies is less than that of babies of average length and of similar age. This fact is illustrated in chart 6. This chart portrays the average values for head size in relation to age for the boys and girls from the metabolism ward in comparison with the subjects of similar ages reported on by the Iowa Child Welfare Research Station.[2] The averages for head circumference of the boys from the metabolism ward correspond more closely with the averages for the girls of the Child Welfare series than with the values for the boys of the latter series. Correspondingly, the body length is similar for the boys from the metabolism ward and for the girls of the Child Welfare series. This comparison has been portrayed in chart 5: The average height in relation to age of the girls of the Child Welfare series has been indicated on this chart through the use of short horizontal bars not elsewhere mentioned.

The foregoing data indicate that head size cannot be interpreted in terms of age unless body length and sex also are taken into consideration. When head size is correlated solely with body length, on the other hand, differences due to sex and to chronologic age tend to disappear

is constancy is illustrated by the data from ston and from Iowa City concerning normal ants which have already been discussed; it also evidenced by the data in chart 7 concerning infants from the metabolism ward. This rt portrays the successive average values for d size in terms of body length, with separate sideration for the infants who have been ignated as immature and those presumably ture at the time of birth, as listed in table 3. r the great majority of the observations the o sets of values are equivalent or identical. ly with a body length of less than 55 cm., head size of the subject personnel made it possible to determine the rate and the constancy of head growth both for individuals and for the group. The results are of interest expressed in relation to either age or body length.

During the first eight months of life the average increase of head circumference closely approximates 9 cm. About 5 cm. of this is gained during the first four months of life. After the eighth month the rate of increase is lessened notably; during the last four months of the first year the circumference increases only 1 to 2 cm. These increment rates are similar for both sexes,

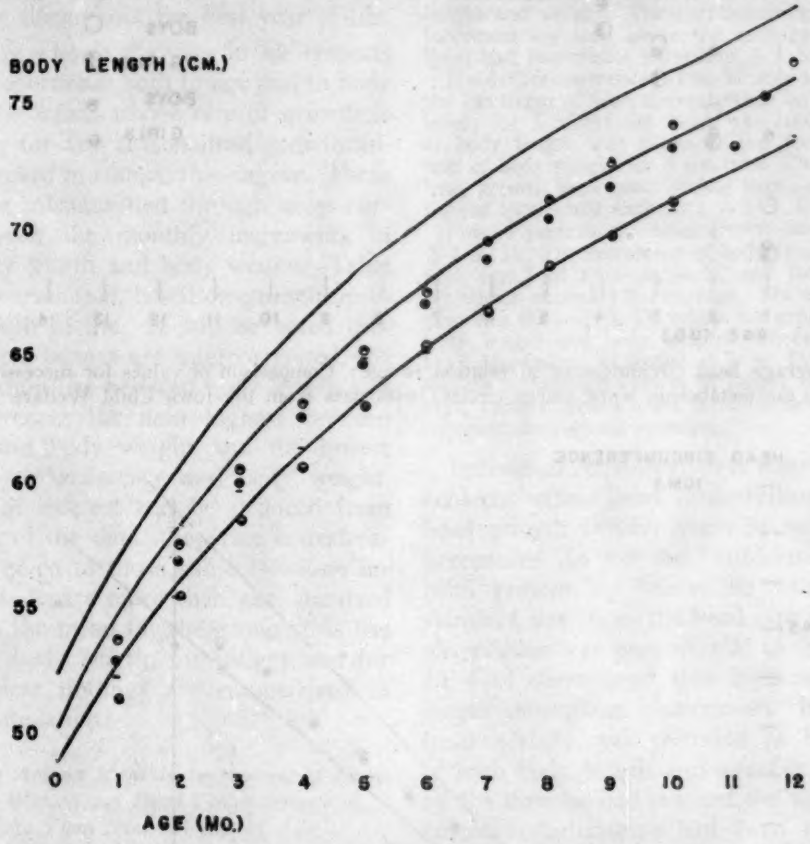


Chart 5.—Average body length of boys from the metabolism ward at different ages, with separate considera- n of those who weighed less than 3,100 Gm. at birth (average 2,800 Gm.). The reference curves are based values for presumably normal infants reported by the Iowa Child Welfare Research Station; they repre- nt the mean with one standard deviation above and one below it. Circles with the lower half shaded rep- sent boys immature at birth; circles with the upper half shaded, the remainder of the group; black circles, erages for the total group.

rresponding to the first two months of life, is ere notable difference between them. Initially ere seems to be a tendency for the heads of ose immature at birth to be disproportionately nall in minor degree. Possibly through correc- on of molding, development of rigidity of the tures or actual accelerated head growth, the ad size overtakes that of the group born with evidences of immaturity and of corresponding ature and maintains identity with it thereafter roughout infancy. Serial measurements of the without regard to initial head size. Few subjects deviate notably from the rates stated; most of the exceptions can be explained on the basis of recognized extraneous factors. Furthermore, the calculated increment rates for head size of the subjects of the Child Welfare study, based wholly on cross-sectional averages, support the stated value.

When the incremental rate of head growth is considered in terms of its relationship to advancing body length rather than to age, the rate of

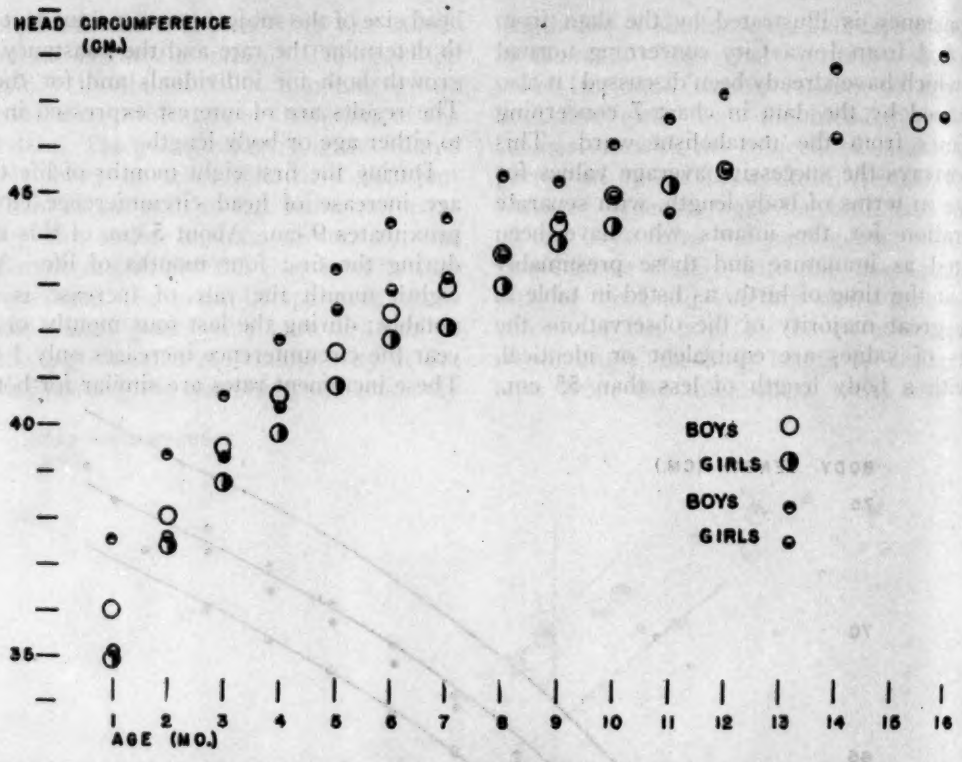


Chart 6.—Average head circumference in relation to age: Comparison of values for successive measurements of subjects from the metabolism ward (large circles) with data from the Iowa Child Welfare Research Station (small circles).

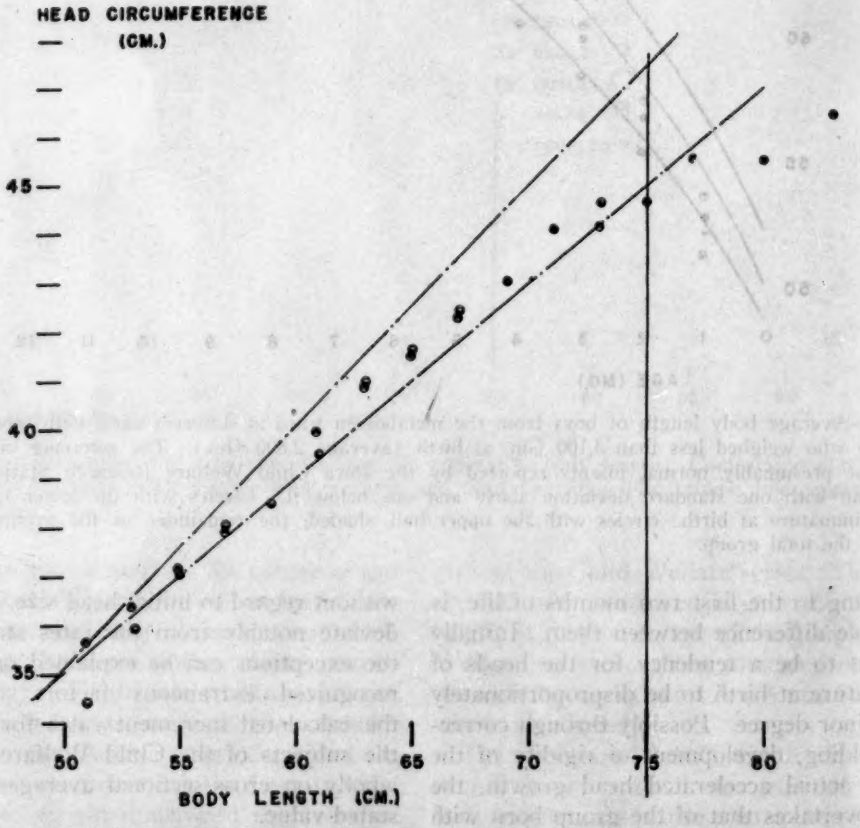


Chart 7.—Head circumference in relation to body length: Comparison of boys from the metabolism ward whose weight at birth was less than 3,100 Gm. (average 2,800 Gm.) with the remainder of the group. The broken lines represent empiric formulas (see text). Circles with the lower half shaded represent boys immature at birth; circles with the upper half shaded, the remainder of the group.

ogression is found to be much more constant. uring the first few weeks, while artefacts due to olding are subsiding, values are less predictable. hen a body length of 52 to 54 cm. has been ained, the increase in head circumference approximates 0.4 cm. for each added centimeter of ight up to a length of 75 cm., or about to the mpletion of the first year of life. As is illustated in chart 7, no notable or characteristic fference in average increment rate can be scerned for prematurely born infants as compared with the remainder of the group. This obrvation holds good with respect to both body ngth and age throughout the first year of life.

For the baby who is thriving in all respects ad size is proportional both to age and to body ngth. For the infant whose rate of growth is owed notably for any reason head growth appears to be checked in comparable degree. These nclusions are substantiated through cross corlations between the monthly increments of ad size, body length and body weight. Table shows such correlation, based on growth up to e eighth month of life. It will be noted that hereas all three factors are intercorrelated, the ghest correlation lies between body length and ad circumference; the next highest between dy length and body weight and the lowest tween head circumference and body weight. ther points of interest can be deduced from oser analysis of the data. Separate consideraon has been given to those subjects whose inement values depart more than one standard viation from the mean for the group; this has en done for body length, for weight and for ad size. These findings are summarized in e following tabulation:

ntercorrelations Among Monthly Increments of Body Length, Body Weight and Head Circumference of Infants Less Than Nine Months of Age

) Average monthly increase in dimension:	Body Length, Cm.	Body Weight, Gm.	Head Circumference, Cm.
Mean	2.53	692.7	1.17
Standard deviation	0.62	184.8	0.37
Standard error of mean	0.06	19.2	0.04

) Correlation coefficients between monthly increments of growth:

Body length and body weight	$+0.5204r$
Body length and head circumference	$+0.7089r$
Body weight and head circumference	$+0.4759r$

) Analysis of outgroups: Those whose individual values lay more than 1 standard deviation (S. D.) away from the mean value for the increment of body length, body weight or head circumference (referred to in the following as substandard or above standard).

Length: Nine values for length increment were subandard. Values for the increment of head size for all these subjects lay at —1 S. D. or lower.

Thirteen values were above standard. All corresponding values for head size were up to the group average; 9 increments exceeded +1 S. D., and 3 others were well above the average.

Weight: Nine values for weight increment were substandard. The values for head increment were similarly substandard for 6 subjects; those for the remainder approximated the average value.

Fourteen subjects had values for weight increment above standard. Eight of these had values for head increment similarly above standard; the increments of 3 others approximated the average, and for the remaining 3 the head increment was less than —1 S. D.

Length and Weight Jointly Considered: The increment values for 2 subjects were substandard in both height and weight. The corresponding values for head increment were similarly below standard.

The values for 7 subjects lay above standard in both height and weight. The corresponding values for head increment lay well above the average for all 7; 6 of them had increments exceeding +1 S. D.

Head Circumference: For 11 subjects the value for the increment of head circumference lay at the —1 S. D. level; for 7 others the value was less. The increment of body length was similarly low for 9 subjects and that of body weight for 6 subjects. Three subjects with head growth increments of less than —1 S. D. had the weight increments exceeding +1 S. D.

For 14 subjects the head growth increment exceeded +1 S. D. The increment of body length was similarly high for 2 of these subjects, and the values for the remainder exceeded the average. Six of the 14 subjects exceeded the +1 S. D. values for growth in head size, body length and body weight. Three subjects whose head increments exceeded +1 S. D. failed to show equally accelerated growth in height and weight; however, in each instance the latter 2 values exceeded the corresponding group averages.

Individual consideration is warranted for those subjects whose head measurements or rates of head growth classify them in one of the outer percentiles. In 5 of the 7 subjects whose rates of head growth lay below the minus 1 level of standard deviation, the head size at the terminal observation was proportional to the body length. In 4 of these head size kept pace with body length throughout observation. In the fifth the head initially was recorded as large in terms of both body length and age (at 1½ months); by the time he had reached the age of 4 months normal relationships had been resumed spontaneously. In 3 of the infants whose head size and length were symmetrically substandard, severe and prolonged infection of the upper respiratory tract and otitic infection had been present. Accurate appraisal of the functional capacity of the brain of the young infant is difficult, even for students who have specialized in such appraisal. The records made by the attendants of these infants are incomplete, and the periods of observation were too short in several instances to permit establishment of relationship or lack of relationship between substandard head size and mental impairment. Two of the 7 infants just discussed, however, were considered by their attendants to be slow of response and of achievement in comparison with other members of the group.

Among those whose head measurements or rates of head growth place them in the upper percentile group, the number of measurements was too few, and the subjects were too young at terminal observations, to rule out errors of measurement or of record. Only 1 subject in that group deserves special mention. This boy was seen first when he was 3 weeks old. His head measured only 34 cm. at that time. The rate of the subsequent growth of the head was steady, and it accelerated both for age and for body length. When he was discharged from observation, at the age of 6 months, the circumference of his head was 45 cm., decidedly large for his age and length. The attendants had recorded in their daily notes that this baby was slow in achievement. At the age of 5 years he was returned to the department outclinic because of "backwardness." At that time his head was significantly small rather than large for his age: 47.5 cm. in circumference. In this instance at least, the deviation from normal head size during infancy probably was evidence of intracranial abnormality, which affected subsequent growth of the brain.

COMMENT

In this study it has been assumed that the greatest circumference of the head provides a fairly reliable index of the capacity of the cranium. This premise is sound except when the head is misshapen either because of molding or from abnormal configuration and when excessive fat or edema is present in the tissues of the scalp. If standard technic is employed, error of measurement is small—much smaller than with measurement of elastic or inconstant structures. For purposes of comparison with subsequent measurements in the determination of the pattern of growth the head circumference is fully as dependable as the body length of the infant and more so than the body weight. Technical errors of measurement result mostly from failure to pass the tape over the greatest circumference. Groups of medical students who previously have not made measurements of infants' heads usually have checked each others' values with a discrepancy of less than 5 mm. Errors in reading the measurement or of recording it are not uncommon. In the current study some recorded values obviously were in error, as when they indicated diminution of head size from earlier measurements. Naturally measurements known to be erroneous have been discarded from consideration; some of those included, however, may also have been in error.

The following standards for progressive head size of infants, based on this study and the reported data of others, are offered. The head circumference at birth of an infant weighing about 3,500 Gm. and 50 cm. in height will approximate 35 cm. An increase of 9 cm. is expected during the first eight months; during the first four months the monthly increase will exceed 1 cm. The rate of increase falls off sharply after the eighth month; from the eighth to the twelfth month the increase is only 1 to 2 cm. If the infant's growth in length is checked, the head growth is affected proportionally; however, subsequent growth in length may lead to resumption of predicted stature, whereas in some small measure the head size may remain less than it otherwise would have been. With average growth in body length, the head circumference at 4 months will be 40 to 42 cm., at 8 months 43 to 44.5 cm. and at a year 45 to 48 cm. In each instance the lower values are more representative for girls or for boys who are not growing as rapidly as the average.

In terms of body length, without regard for sex or age, once neonatal distortions have been corrected the head circumference usually will fall between two empirically derived formulas: $Hd = 0.4L + 15$ and $Hd = 0.5L + 10$, in which Hd refers to head circumference and L to length, each expressed in centimeters. The well-fed, healthy infant will conform more closely to the second formula or tend to surpass it. The first formula fits better the infant who is chronically ill, underfed or handicapped by other extrinsic or intrinsic factors; it represents suboptimal growth of head or of body or of both. The slower the rate of head growth, the sooner such growth will be terminated and the smaller the ultimate head size.[5]

From the formula just given, the circumference of the head increases about 0.4 cm. for each added centimeter of body length up to a length of 75 cm., or approximately through the first year of life. Through individualization of the data for those infants whose measurements fall below or beyond the limits stated, the clinician may detect evidences of abnormal growth of the intracranial contents earlier than otherwise would be possible.

Aside from consideration of relationship between size of head and length of body, the significance of deviant body length should not be overlooked. Review of the data used in this

5. H. Bakwin and R. M. Bakwin (J. Clin. Investigation **10**:377 [June] 1931) derived the formula $Hd = 0.458L + 11.8$ from measurements of pooled data on groups of favored and of less favored infants. The published data do not permit analysis of deviations with respect to rate of growth in the contrasted groups of infants.

dy has brought emphasis to the concept that rate of growth even among the average infant ulation may be conditioned favorably or unorably by commonplace environmental fac-. If the growth of the head is conditioned the same set of factors, as the data suggest, fact may be significant in determining the ensions of the adult head. The stimulus for wth of the cranium is related to the early inase in volume of the central nervous system. ause of the precocity of the growth of the in and the subsidence of its propensities ard growth after infancy, factors impeding d growth during infancy may well lead to perent limitation of head size. By inference bulk of the brain would be affected in parallel ree. What effect this circumstance might e on the subsequent capacity of the central vous system for functioning in an optimum nner or for resisting insult is a matter for ious speculation.

CONCLUSIONS

During infancy the size of the head is directly ated to the length of the body. A simple forla can be used to predict the average head cumference for the infant whose body length s not exceed 75 cm., without regard for age sex. Through proper appraisal of head size d of body length the probability of abnormality growth of the intracranial contents can be imated.

DISCUSSION

Dr. Harold C. Stuart, Boston: I could not say, hout referring to my data, whether Dr. Boyd's asption is correct. My recollection is that after the t three months it would be correct; that is, the ldren who stood high in the distribution in body gth usually stood high also in head size. But there a number of factors operative in the early months ich may alter this picture. In connection with the ice of a base line, I am inclined to believe that it uld be better to take the size of the head at 2 weeks of rather than at birth. This would allow neonatal justments to take place and would provide a meaement which would more accurately represent the of the head. One does see, of course, small heads long babies and vice versa, but as a general prinle I should have expected the small heads to become atively larger and the large ones relatively smaller ring the first two years.

Just what unusual size or rate of growth of the head ans in any given case is often impossible to say, but is important to realize that a number of factors mbine to determine the rate of growth of the head. ith Boyd and Scammon took monthly tracings of fontanels of infants until complete closure. These cings showed that the fontanels do not progress iformly toward closure but vary in size from period period. I believe that they considered this fluctuan in size to be in some way related to diet or to trition: the net result at a given time of the conflictg forces of enlargement of the skull due to growth of brain and ossification of the membranous bones surrounding the fontanels.

There are one or two points I should like to emphasize in connection with the use of measurements of the size of the head. A single measurement of the head is of little help, for the actual measurement is not outside normal limits unless the head is so large that you know it is large, or so small that you know it is small, from inspection alone. If one is going to use measurement of the head as a means of recording the rate of growth of the head, one must adopt a definite technic and follow it consistently. It does not have to be any particular technic unless one is going to use some particular set of standards, but measurements of the head plot a very irregular curve unless carefully taken. I believe that the length and the breadth of the head are actually a little more accurate as measured by most persons than is the circumference of the head, but not enough so to justify changing to these measurements if one is in the habit of taking the circumference.

If you are going to include a measurement of the head in your battery of measurements of growth—and I believe it is desirable to do so in the first two years at least—take the initial measurement at about 2 weeks of age, after the early adjustments have taken place, and be sure to take the measurement in the same way at succeeding periods.

Dr. Béla Schick, New York: I am teaching my staff a simple and practical relationship which exists between the circumference of the head and the sitting height. The differences between the figures for total height are lessened by omitting the lower extremities.

Circumference of the head and sitting height are equal during the first ten months of life. Each of these measures increases about 1 cm. per month. Greater differences between the two measures indicate either microcephalus or hydrocephalus.

I should like to ask Dr. Boyd whether he measured the sitting height of the children. Thus he would be able to compare my statement with his results.

Dr. Harry Bakwin, New York: Dr. Boyd suggests that the circumference of the head and the height of the body be correlated, and Dr. Schick suggests that sitting height be used. It would seem to me that the simplest and most useful base line is age. The circumference of the head is closely related to age. The standard deviation of the circumference of the head according to age is only about 3 to 4 per cent. It is less variable in relation to age than are any of the other external dimensions of the body.

I should like to ask Dr. Boyd what the advantage is of correlating the circumference of the head and the total height of the body.

Dr. Harold C. Stuart, Boston: May I say one more word in connection with the use of standards of reference in the interpretation of measurements, for only by proper use of suitable standards can measurements be of practical value. I am in the habit of using tables which give various percentiles, whereas Dr. Bakwin uses mean and standard deviations. I do not feel that it makes any difference which one uses, but it does matter how one uses them. I find the percentile rank into which a child's measurement of length or height falls and then compare the rank obtained for all other measurements with that rank. By that means it is easy to see at a glance whether the measurement of the head falls in a higher or a lower rank than other measurements and if so to tell how unusual it may be. I simply look at all measurements from this point of view, and if any one is out of line with the others I follow its progress with growth and try to recognize

any significance which may attach to an unusual course of events. By having the percentile ranks of several measurements in front of him, a person can compare them and recognize at a glance unusual features of size or proportions or rate of growth of the body as a whole or of any part which has been measured. In other words, I quickly forget the measurements themselves and consider only what relationship they bear to the range of measurements for normal children of the same age and sex and to the measurements found at previous examinations.

DR. J. D. BOYD, Iowa City: Anthropometrists long have recognized the interrelationship among measurements of various parts of the body. I had available serial measurements of weight, crown-heel length, crown-rump length and breadth and length as well as circumference of the head for the subjects studied. No doubt, as Dr. Schick suggested, crown-rump length could be used to advantage as a basis for estimation of the normality of the size of the head. Such measurement is obtained readily and is less subject to technical error than is measurement of crown-heel length. I chose, however, to use crown-heel length, because it is a measurement of greater meaning to the parent and to the physician and because it portrays better the growth of the entire skeleton. The circumference of the head seemed to offer a better index of intracranial volume than the width or the length of the head. The last two measurements require the use of calipers; moreover, their individual pattern of increase is less constant than is that of the circumference of the head. When the eye reveals dissymmetry of the head, judgment should be used as to the acceptability of any linear measurements in the estimation of the volume of the head. I agree with Dr. Stuart that measurements of the circumference of the head made during the first few weeks of life may be misleading because of cranial distortion. Mention should be made of the common practice of estimating the normality of the size of the head by comparison with the size of the chest. Measurement of the chest is subject to marked technical error, which is dependent on a variety of factors. There seems little reason to continue estimation of the normality of the size of the head in such terms if accuracy of appraisal is desired.

In reply to Dr. Bakwin's comment, I agree that one can use age rather than length of the body in estimating the predicted size of the head when considering the infant of normal proportions. The sex of the infant must be considered in the interpretation, however, where sex differences disappear when the length of the body is the basis for comparison. Often the clinician is concerned with an infant of normal proportions: he wants to know the appropriateness of the observed size of the head for an infant who has failed to grow commensurately with his age. In such an infant or in an infant who has been born prematurely age may be a poor index of the size of the head. The size of the head as related to age serves to indicate the status of the head as to total growth; when it is expressed in terms of the length of the body the same information may be gained, with additional insight as to symmetry of growth.

This study has been designed to serve the clinician rather than the anthropometrist. Many of the measurements made routinely by the anthropometrist have little apparent importance for the clinician. I believe that if one makes and records careful serial measurements of the weight and the length of the body and the circumference of the head together with the exact age, one will be in a position to appraise with fair accuracy the normality of the pattern of the infant's growth without recourse to further anthropometric data.

MMUNITY TO TETANUS INDUCED BY A THIRD DOSE OF TOXOID FOUR YEARS AFTER BASIC IMMUNIZATION

BASED ON A STUDY OF TWENTY-FIVE ALLERGIC CHILDREN

M. MURRAY PESHKIN, M.D.

NEW YORK

The increased interest of the medical profes- in active immunization against tetanus has n due to the negligible incidence of tetanus ong the immunized soldiers engaged in the sent world conflict. Of special practical im- tance is the degree of immunity induced by "booster" dose of tetanus toxoid in relation the interval between basic immunization and inistration of the "booster" dose.

In my first report,[1] on the appearance of anus antitoxin in the blood serum following injections of 0.5 cc. each of combined alum- cipitated diphtheria and tetanus toxoids, it s shown that 94.6 per cent of a group of 186 rgic children acquired a protective level of anus antitoxin when 0.01 American unit of itoxin per cubic centimeter of blood was con- ered a critical level. In a second study[2] a rd, or "booster," injection, of 0.5 cc. of the nbined toxoids, was given to 65 of the same children. Nine of these 65 children had failed attain adequate immunity after receiving the st two doses of the combined toxoids. The oster" dose was administered three to fifteen nths after completion of basic immunization. two subsequent communications[3] I reported the effect of "booster" injections of 0.5 cc. of combined diphtheria and tetanus toxoids or tetanus toxoid alone given respectively to 31 and 38 other children of those described in the first report two and three years after completion of the basic immunization. The "booster" dose of toxoid stimulated the production, in all instances, of an adequate titer for tetanus antitoxin. Moreover, the "booster" dose of toxoid given three years after completion of basic immunization was always followed by a tetanus antitoxin titer which was higher and lasted much longer than that which developed after basic immunization or after the "booster" dose of toxoid injected three to fifteen months and two years after basic immunization.

In this communication I am reporting the results of a comparable study on the degree and duration of immunity after a "booster" injection of 0.5 cc. of tetanus alum-precipitated toxoid, given four years after basic immunization to 25 of the 186 children used in the study described in the first report. All of these 25 children had attained adequate immunity after receiving the two initial doses of the combined toxoids.

MATERIALS AND METHODS

The total number of samples of blood titrated for tetanus antitoxin from the 25 allergic children after the "booster" injection was 101. The dose consisted of 0.5 cc. of alum-precipitated tetanus toxoid; it was administered four years after completion of the basic immunization with two doses of the combined diphtheria and tetanus alum-precipitated toxoids. One hundred and eighty-five samples of blood had been titrated after the first two injections.

TETANUS ANTITOXIN TITERS AFTER THE SECOND AND AFTER THE THIRD, OR "BOOSTER," DOSE OF ALUM-PRECIPITATED TOXOID

Examination of table 1 shows that for 22 patients titers determined after administration of

F. G. Jones, of the Lilly Research Laboratories, e technical assistance in this study.

From the Children's Allergy Clinic, Mount Sinai spital: Dr. Murray H. Bass, Director of the Department of Pediatrics.

The preparations of combined alum-precipitated diphria and tetanus toxoids and alum-precipitated tetanus oid used in this study were furnished by Eli Lilly Co.

1. Peshkin, M. M.: Immunity to Tetanus Induced Combined Alum-Precipitated Diphtheria and Tetanus xoids, Based on a Study of One Hundred and Eighty- Allergic Children, Am. J. Dis. Child. **62**:9 (July) 1.

2. Peshkin, M. M.: Immunity to Tetanus Induced Combined Alum-Precipitated Diphtheria and Tetanus xoids, Based on a Study of Sixty-Five Allergic Chil- n Given a Third or "Repeat" Dose, Am. J. Dis. ild. **62**:309 (Aug.) 1941.

3. Peshkin, M. M.: (*a*) Immunity to Tetanus Induced by a Third Dose of Toxoid Two Years After sic Immunization, Based on a Study of Thirty-One ergic Children, Am. J. Dis. Child. **65**:873 (June) 43; (*b*) Immunity to Tetanus Induced by a Third Dose of Toxoid Three Years After Basic Immunization, Based on a Study of Thirty-Eight Allergic Children, ibid. **67**:22 (Jan.) 1944.

the second dose of toxoid were available for comparison with the 51 which were obtained at intervals of one week to one year after administration of the third, or "booster," dose. The values for antitoxin after administration of the third dose of toxoid were higher in 48 of the 51 instances and the same in 3. After administration of the third dose the average antitoxin value was fifteen times the average corresponding titer after administration of the second dose.

From the data on 17 children it was found that when initial tests, made within two months after administration of the "booster" dose, showed the antitoxin values to be higher after the third dose was given than after administration of the second, then tests performed one year later revealed not only that the drop in titer was retarded after the third dose was given but that the antitoxin values were distinctly higher (a ratio of 6.7 to 1, or an average antitoxin titer of 0.353 unit as compared with 0.053 unit).

ANTITOXIN TITER WITHIN ONE MONTH AFTER ADMINISTRATION OF THE "BOOSTER" DOSE OF TOXOID

Data from which to determine the effect of the "booster" dose of toxoid on the antitoxin response, with reference to the antitoxin titers obtained after the second dose of toxoid was given and immediately preceding administration of the third, were available for 25 patients. These patients were divided into two groups.

The first group consisted of 11 children who had adequate antitoxin levels soon after administration of the second dose of toxoid but whose titers some months later dropped to the levels of 0.003 and —0.003 unit per cubic centimeter of serum. After the third dose of toxoid was given the titers of these patients ranged from 0.2 unit to 5 units, with an average titer of 1.21 units. The titers were increased from 67 to 1,667 times their original levels; on the average, the titers were increased to 342 times their original levels.

The other group consisted of 14 patients who also had adequate antitoxin responses soon after the second dose of toxoid was given but whose subsequent titers, obtained four years later, though lowered, remained above the critical level for protection against tetanus. After the patients received the third dose of toxoid the titers for 13 of them ranged from 1 to 10 units of antitoxin, with an average titer of 3.692 units as compared with an average titer of 0.051 immediately preceding administration of the dose. The titers were increased to from 5 to 588 times their original levels; on the average they were increased to 148 times their original levels. (The data for the remaining patient—no. 24, table —in this group were omitted in computing t averages because of the unusually high antito titer of 30 units obtained within one month af injection of the third dose of toxoid).

The data obtained in this study corrobora the observation, reported in previous commu cations, that the antitoxin levels attained wit one month after administration of a third, "booster," dose of toxoid vary with those pr ent immediately preceding the "booster" do When the titer dropped to the inadequate le (0.003 unit of antitoxin) after having been a quate (0.01 unit or more of antitoxin), then t average titer following administration of a thi dose rose to 1.21 units of antitoxin. When t titer was always maintained at the adequate le after administration of two doses of toxoid, th the titer following administration of a third do reached the highest value, with an average 3.692 units of antitoxin. For the patients whom the titer had dropped to an inadequate le a third dose of toxoid given three to fifte months,[2] two years [3a] or three years [3b] after t second injection induced antitoxin values whi bore the following respective ratios to those r ported in the present study: 1.4:1, 0.9:1 a 1.1:1. For those patients who had maintain adequate titers the antitoxin values obtain when the third dose of toxoid was given aft four years were higher than the values obtain by giving it three to fifteen months or two yea after the second injection, and the same was tr of the values obtained by giving it three yea after the second injection. The ratios we 3.3:1, 1.5:1 and 1.1:1 respectively.

TIME OF OCCURRENCE OF THE MAXIMUM RESPONSE TO ANTITOXIN

To determine the maximum response to anti toxin following administration of the "booster dose of toxoid, two or three tests were done o each patient within two months after the inje tion. Seven days after the third dose was give the antitoxin titers per cubic centimeter of seru were as follows: 0.2 unit, 6 children; 0.5 unit, children; 1 unit, 7 children; 2 units, 5 children 5 units, 2 children; 10 units, 1 child, and 20 unit 1 child. These results indicate that adequa levels of tetanus antitoxin (0.2 unit or more were obtained seven days after injection of "booster" dose of 0.5 cc. of the alum-precip tated tetanus toxoid in all of the children wh were treated four years after completion of bas immunization. For 8 of the patients the anti toxin titer was at its maximum seven days aft administration of the toxoid, while for the re maining 17 patients the titer was at its maxim

hin one month. For the former group of 8 dren the maximum antitoxin levels were as ows: 0.2 unit, 1 child; 0.5 unit, 1 child; from o 5 units, 5 children, and 10 units, 1 child. the latter group of 17 children the maximum itoxin levels were as follows: 1 unit, 6 chil- n; from 2 to 5 units, 10 children, and 30 ts, 1 child.

The foregoing observations confirm the results orded in the three earlier studies on compar- e groups of children who received the "boost- injection of toxoid three to fifteen months, o years and three years after basic immuniza- n. However, higher maximum antitoxin values e obtained after the third dose of toxoid for majority of the children receiving the third e three years after basic immunization. For majority of the children of the present series her maximum antitoxin values were compar- e to those obtained for the group of children eiving the third dose two years after basic munization. In the group of children receiv- the third dose within three to fifteen months r basic immunization titers which were ade- te for protection were attained by all of the dren within one month after they received "booster" dose of toxoid. However, the ups of children given the third dose of toxoid o, three and four years after basic immuniza- n attained adequate titers within seven days. the majority of the children in the first three ups the antitoxin titers were at maximum el on the seventh day after the "booster" dose toxoid was administered, while for the ma- ity of the children in the present series the itoxin titers were at maximum level one nth later. For every patient the maximum s reached within two months or less.

ATE OF DECLINE OF THE ANTITOXIN TITER TO AN INADEQUATE LEVEL

In the preceding study 36 children were given booster" dose of toxoid three years after basic munization and were available for testing one r after the "booster" dose was administered. e percentage of children in whom there was a p in the level of antitoxin from an adequate an inadequate level (0.003 unit per cubic centi- ter of serum) and the number of months re- red for this change were shown in a chart. r purposes of contrast, there were also shown the chart one curve constructed from the data the group of 30 children who received the rd dose of combined toxoids or of tetanus oid alone two years after the second injection d another for the group of 59 children who eived the third dose three to fifteen months r basic immunization.

Examination of the chart showed that when the "booster" dose of toxoid was given three years after basic immunization 100 per cent of the 36 children maintained adequate immunity for a year. In contrast, of the 30 children given the "booster" dose two years after basic immunization, 100 per cent maintained adequate immunity for ten months and only 97 per cent for a year. Of the 59 children given the "booster" dose three to fifteen months after basic immunization, 100 per cent maintained adequate immunity for two months, 98 per cent for six months, 97 per cent for ten months and only 93 per cent for a year.

All of the 25 children tested in the present study acquired adequate levels of tetanus antitoxin within one month after receiving the "booster" dose of toxoid. Seventeen children were available for testing one year after the "booster" dose was administered. One hundred per cent of these 17 children maintained adequate immunity for a year. This is comparable to the results for the group given the "booster" dose three years after basic immunization. This fact is of importance when one considers that there is no cutaneous test available for the determination of immunity to tetanus and that titration of blood serum for tetanus antitoxin is not a pracical laboratory procedure. Moreover, the average antitoxin levels one year after the "booster" dose for the four groups of children were as follows: 0.24 unit of antitoxin per cubic centimeter of blood serum for the group of children given the "booster" dose of toxoid four years after the basic immunization (the data on 1 patient—no. 24, table 1—were purposely omitted when the average titer was calculated, since inclusion of a high titer would cause distortion of the average); 0.373 unit of antitoxin for the group of children given the "booster" dose of toxoid three years after basic immunization; 0.298 unit of antitoxin for the group of children given the "booster" dose of toxoid two years after basic immunization, and 0.083 unit for the group of children administered the "booster" dose three to fifteen months after basic immunization.

INITIAL MAXIMUM ANTITOXIN LEVEL COMPARED WITH THE LEVELS ONE YEAR AND LESS THAN ONE YEAR AFTER THE THIRD DOSE OF TOXOID

Seventeen of the 25 children were available for retesting one year after receiving the "booster" dose of toxoid. In table 2 the maximum antitoxin titers obtained within the first month and the titers obtained one year after administration of toxoid (or less than one year in those cases in which the titer dropped to the inade-

TABLE 1.—*Tetanus Antitoxin Titers of 25 Allergic Children Obtained After the Second Injection of Combined Alum-Precipitated Diphtheria and Tetanus Toxoids (Basic Immunization) and After the Third, or "Booster," Injection of Tetanus Alum-Precipitated Toxoid Alone*

Patient Number	Age, Yr.	Antitoxin Titer, Units per Cc. of Serum After Injections of Toxoid: After Basic Immunization 1 Wk.	1 Mo.	2 Mo.	6 Mo.	1 Yr.	2 Yr.	3 Yr.	4 Yr.	After Booster Dose 1 Wk.	1 Mo.	2 Mo.	6 Mo.
1	11	..	0.1	0.05	0.01	0.003	0.003	—0.003**	—0.003	0.2	1.0	...	...
2	14	..	...	0.017†	....	0.003*	0.003	.....	—0.003	0.2	0.2	0.1	...
3	4	..	0.1	....	0.003	0.003††	.....	—0.003	—0.003‡‡	0.2	1.0	0.5	0.5
4	5	..	...	0.2	0.017	0.017§§	0.003	0.003	—0.003‡‡	2.0	2.0	2.0	0.2
5	6	..	...	0.2	0.1	0.003	0.003	0.003	—0.003	5.0	2.0	2.0	0.1
6	5	..	0.1	0.05	0.003	0.003	0.003	0.003	0.003	0.2	1.0	0.5	0.2
7	4	..	0.1	....	0.01	0.01††	0.01**	0.003‡‡	0.003	0.2	1.0	0.5	0.2
8	4	..	0.2	....	0.003	0.003	0.003**	0.003	0.003	0.2	1.0	...	...
9	11	..	...	....	0.05	0.003	.....	.....	0.003	0.5	0.5	0.5	...
10	11	..	...	0.2	....	.....	.....	.....	0.003	1.0	2.0	2.0	...
11	9	..	...	0.2	0.017	0.01	0.01	0.003	0.003	1.0	2.0	1.0	...
12	5	..	...	0.5	0.1	0.1	0.05**	0.017‡‡	0.017	1.0	2.0	...	...
13	4	..	...	0.1†	0.05	0.05	0.05**	0.05‡‡	0.017	1.0	2.0	2.0	0.5
14	5	..	...	....	0.05	0.017††	0.017	0.017	0.017	1.0	2.0	1.0	0.2
15	6	..	...	....	0.017	0.017	0.01	0.017**	0.017	2.0	1.0	0.2	0.1
16	7	..	0.5	....	0.1	0.05††	0.1	0.05	0.017‡‡	2.0	5.0	5.0	2.0
17	4	..	...	0.2	0.05	0.05	0.017††	0.017**	0.017‡‡	5.0	2.0	...	...
18	11	..	0.5	....	0.05	0.2§	0.05**	0.05	0.017	10.0	5.0	2.0	...
19	7	..	...	2.0	0.1	0.05††	0.05**	0.05‡‡	0.05	0.5	2.0	2.0	0.5
20	6	1.0	1.0	...	0.05	0.05	0.017††	0.05**	0.05‡‡	1.0	5.0	2.0	...
21	7	...	...	0.5	0.1	0.05††	0.05	0.05	0.05‡‡	2.0	2.0	2.0	0.2
22	8	...	...	0.2	0.1‡	0.2††	0.2**	0.2	0.1	1.0	5.0	...	...
23	6	...	0.5	0.2	0.1	.....	0.2	0.1	0.1	2.0	5.0	2.0	0.5
24	3	...	...	0.5†	0.2	0.1††	0.2**	0.1‡‡	0.1	20.0	30.0	10.0	5.0
25	6	0.2	...	0.2	0.05	0.05	0.1**	0.1‡‡	0.2	0.5	1.0	0.5	0.5

† Titer at 4 months.
* Titer the same at 9 months.
‡ Titer the same at 10 months.
§ Titer 0.10 unit at 10 months.
†† Titer the same at 1½ years.
** Titer the same at 2½ years.
‡‡ Titer the same at 3½ years.
§§ Titer 0.01 at 1½ years.

TABLE 2.—*Maximum Tetanus Antitoxin Titer of Allergic Children and Titers Obtained Less Than One Year (When the Titer Dropped to 0.003 Unit of Antitoxin per Cc. of Blood Serum) and One Year After the Third, or "Booster," Injection of Alum-Precipitated Tetanus Toxoid* *

Maximum Antitoxin Titer	Patients No.	%	Antitoxin Titer, Units per Cc. of Serum, After Injections of Toxoid: Less Than 1 Yr. 0.003† No.	%	One Year 0.008 No.	%	0.01‡ No.	%	0.05 No.	%	0.1 No.	%	0.2 No.	%	0.5 No.	%	1 or More No.	%	0.01 or No.
Booster Dose Given Between 3 and 15 Months After Basic Immunization																				
0.01‡	1	2	1	100	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
0.1	3	5	1	33	0	0	2	67	0	0	0	0	0	0	0	0	0	0	2	
0.2	9	16	0	0	2	22	5	55	1	11	1	11	0	0	0	0	0	0	7	
0.5	18	32	0	0	0	0	8	45	4	22	6	33	0	0	0	0	0	0	18	
1.0 or more	25	45	0	0	0	0	6	24	4	16	7	28	7	28	1	4	0	0	25	
Total	56	100	2	4	2	4	21	37	9	16	14	25	7	12	1	2	0	0	52	
Booster Dose Given 2 Years After Basic Immunization																				
0.5	2	7	0	0	0	0	0	0	0	0	2	100	0	0	0	0	0	0	2	
1.0 or more	28	93	0	0	1	4	0	0	3	11	4	14	10	36	8	29	2	7	27	
Total	30	100	0	0	1	3	0	0	3	10	6	20	10	33	8	27	2	7	29	
Booster Dose Given 3 Years After Basic Immunization																				
0.2	1	3	0	0	0	0	1	100	0	0	0	0	0	0	0	0	0	0	1	
0.5	6	17	0	0	0	0	1	17	3	50	2	33	0	0	0	0	0	0	6	
1.0 or more	29	80	0	0	0	0	1	3	4	14	4	14	8	28	7	24	5	17	29	
Total	36	100	0	0	0	0	3	8	7	20	6	17	8	22	7	19	5	14	36	
Booster Dose Given 4 Years After Basic Immunization																				
1.0 or more	17	100	0	0	0	0	0	0	2	12	1	6	9	53	4	23	1	6	17	
Total	17	100	0	0	0	0	0	0	2	12	1	6	9	53	4	23	1	6	17	

* Data are included only for those children whose titrations were performed within forty days after the injection of toxoid. Data for the 56 children given a "booster" dose of toxoid between three and fifteen months after basic immunization, for the 30 children given injections two years after basic immunization and for the 36 children given injections three years after basic immunization were obtained in previous studies (footnotes 2 and 3). Twelve of the 30 children who received a "booster" dose two years after basic immunization, 15 of the 36 children given injections three years after basic immunization and all of the children given injections four years after basic immunization were given tetanus alum-precipitated toxoid alone. All percentages are approximate.

† Data for patients with an antitoxin titer of 0.003 unit less than one year after the "booster" dose are included because such titers persist at this level for at least two years.

‡ Patients with antitoxin levels of 0.017 and 0.025 unit are grouped with those having a titer of 0.01 unit.

te level earlier than this) are compared. The le also gives the comparable data for the chil- n who received the third dose of toxoid three ifteen months after basic immunization and those who received it two and three years r basic immunization.

Four per cent of the 56 children who received booster" dose of toxoid three to fifteen months r the basic immunization and none of those o received it two, three and four years after basic immunization showed a drop in titer m a level adequate for protection to an inade- te level in less than one year.

At the end of one year the level of antitoxin maintained at 0.1 unit per cubic centimeter erum in 88 per cent of the children who were en the third injection four years after they re- ved the second dose (group A), in 72 per cent hose who were given the "booster" dose three rs after they received the second dose (group in 87 per cent of those who received the in- ion two years after the basic immunization oup C) and in only 39 per cent of the children o were given the "booster" dose three to fifteen nths after the basic immunization (group D). e ratios of 1.2:1 between groups A and B, between groups A and C and 2.2:1 between ups A and D are largely results of the dif- nce in the initial maximum titers. One hun- d per cent of the children of groups A and C ompared with 97 per cent of the children of up B and 77 per cent of the children of group ad maximum antitoxin titers of 0.5 unit or e of antitoxin. Thus, it can be observed that antitoxin titer had dropped to an inadequate l in one year or less in 7 per cent of the up of 56 children who were given the "boost- dose of toxoid three to fifteen months after basic immunization, in 3 per cent of the group 0 children who were given the "booster" in- ion two years after the basic immunization in none of the respective groups of 36 and hildren who were given the "booster" injec- three and four years after the basic immuni- on.

REACTION AND SENSITIVITY

or all of the 25 children in the present series "booster" dose consisted of alum-precipitated nus toxoid. None of these children showed a ile or a systemic allergic reaction after the ction. However, 3 children experienced a lerate and 3 a severe local reaction.

hus far in my experience febrile and systemic gic reactions have occurred only after in- ion of the combined alum-precipitated diph- ia and tetanus toxoids. The incidence of ile reactions in the children given the "boost- dose three years after basic immunization, 33 per cent, contrasts with the incidence of 22 per cent among the children given combined toxoids two years after the basic immunization and with the incidence of 3 per cent among the children given combined toxoids three to fifteen months after the basic immunization. Two allergic systemic reactions were encountered in the active immunization of 186 allergic children who had had two to five doses of combined toxoids, including 61 children who had had a third dose of alum-precipitated tetanus toxoid alone, that is, in a total of 534 injections (an incidence of 0.37 per cent).

The scratch tests, performed with undiluted combined diphtheria and tetanus alum-precipitated toxoids and with tetanus alum-precipitated toxoid alone, did not produce positive reactions, moderate or delayed, in any of the children observed in this study. The tests were performed prior to administration of the third dose of toxoid and from one to six months (or an average of two and one-half months) after administration.

In view of the aforementioned findings and in order to avoid the accident of a febrile or systemic allergic reaction following a "booster" dose of combined alum-precipitated diphtheria and tetanus toxoids, it is advisable to employ alum-precipitated tetanus toxoid only, especially when the "booster" dose is given to older children two or more years after completion of the basic immunization with two doses of the combined toxoids.

COMMENT

The capacity to respond with high antitoxin levels to a stimulating, or "booster," dose of alum-precipitated tetanus toxoid given four years after basic immunization with two 0.5 cc. doses of the combined alum-precipitated diphtheria and tetanus toxoids is clearly shown by the results obtained with 25 children. It is believed that the present study represents the only one thus far reported in the literature.

The results obtained in the present study confirm the observation, reported in four previous studies, that the intervals between administration of the first and the second basic dose of the combined toxoids and between administration of the second and the third, or "booster," dose have a direct influence on the response to the antitoxin. The longer the intervals (up to three years) between the injections of toxoid the higher the response to the antitoxin. In the second study [2] the second and third doses of toxoid were given at intervals of three to fifteen months, in the third study [3a] at an interval of two years, in the fourth study [3b] at an interval of three years and in the present study at an interval of four years. The vast majority of the children in the

fourth group responded with the highest antitoxin values. The highest antitoxin values reported for the children in the present study approximately parallel the highest titers recorded for the children in the third group. In the light of all of these studies I believe that an interval of five or more years between the basic immunization and administration of the "booster" dose of toxoid would result in a highly protective response to antitoxin.

SUMMARY AND CONCLUSIONS

Four years after completion of the basic immunization with two 0.5 cc. doses of combined alum-precipitated diphtheria and tetanus toxoids 25 allergic children were given a third, or "booster," dose of 0.5 cc. of alum-precipitated tetanus toxoid alone. The level of antitoxin was determined at various intervals after the second and after the third dose of toxoids, and the respective totals of 185 and 101 specimens of blood serum were titrated for tetanus antitoxin. No febrile or systemic allergic reaction (hay fever, urticaria or asthma) occurred.

Prior to administration of the "booster" dose and from one to six months (an average of two and one-half months) thereafter reactions to scratch tests with undiluted combined alum-precipitated diphtheria and tetanus toxoids and with alum-precipitated tetanus toxoid alone gave negative results.

The injection of a third, or "booster," dose alum-precipitated tetanus toxoid into aller children four years after completion of ba immunization with two doses of combined alu precipitated diphtheria and tetanus toxoids w followed within one month in all instances by adequate tetanus antitoxin titer. This titer w always higher and lasted for a much longer per than that which followed basic immunization well as that which followed administration o "booster" dose of toxoid three to fifteen mon after basic immunization. However, this titer w comparable to the maximum antitoxin respo following a "booster" dose of toxoid given t years after the basic immunization. The highe antitoxin values thus far obtained followed "booster" dose of toxoid injected three ye after the basic immunization. The antitoxin ti obtained on the seventh day after administrati of a "booster" dose of toxoid given four ye after the basic immunization was always a quate, and it was at its maximum one mon later in the majority of instances.

Finally, when a child has had basic immu zation with two doses of combined alum-preci tated diphtheria and tetanus toxoids and a thi injection of toxoid is administered two to fo years later, then alum-precipitated tetanus toxo alone should be used for the third injection order to keep local and systemic allergic re tions at the minimum level.

450 West End Avenue.

CHANGES IN THE VIBRATORY SENSE OF PATIENTS WITH POLIOMYELITIS

AS MEASURED BY THE PALLESTHESIOMETER

WILLIAM O. FROHRING, B.S.; PAUL M. KOHN, M.D.; JAMES F. BOSMA, M.D., AND JOHN A. TOOMEY, M.D.

CLEVELAND

In poliomyelitis there are obvious pathologic anges in the posterior as well as in the an- ior area of the spinal cord and in the cells of sensory ganglions outside the cord. Never- eless, after the first attack of pain, which is arly segmental, sensation seems uninvolved. may be that sensory involvement has not en demonstrated clinically because the ordi- ry methods of measurement at the disposal of ysicians are too gross to detect minute quanti- ive or qualitative changes in sensation. A cently developed pallesthesiometer has been und satisfactory in other neurologic studies, d it was decided to test patients with polio- elitis by means of this instrument.

METHOD

Mechanism of the Instrument.—The pallesthesiometer ntitatively measures the vibratory sense. The one d in the present studies has been described in detail an article published elsewhere.[1] It consists of a disk or tton which vibrates in a vertical plane. The ampli- de of vibration of the disk is controlled by two levers on dials, each of which is divided into 100 equal its; thus there is a total of 200 dial units. The in- ument is calibrated by determining the voltage across terminals of the vibrator for each of the 200 dial ttings. With the particular vibrator used in the sent experiments, it can be shown that the ampli- de of vibration is proportional to the square of the ving current and, hence, to the square of the applied ltage. It becomes obvious, then, that a comparison two readings made in terms of the square of the plied voltage will give a ratio directly proportional to amplitudes of vibration. For example, subject A ght respond at the point of v^2 100 and subject B at point of v^2 200. We could then say that for sub- t B the amplitude of vibration is twice as great as for subject A. This method allows a more fundamental comparison of data than that made possible by the use of arbitrary dial units.

With this method of calibration, 0 on the dial corresponds to 4.9 volts, or v^2 24.01, and 200 is equivalent to 45.2 volts, or v^2 2,043.

Determination of the Vibratory Thresholds.—To determine the threshold of the vibratory sense at various points on the surface of the body, the disk was applied locally, and the operator gradually increased the amplitude of vibration until the subject first became aware of the vibration. The average dial reading at this point is called the "threshold value." This procedure was checked several times, a rest period being allowed between stimulations. The "threshold value" varied in different persons, in different points on the same person and to a much lower degree on the same point on the same person on different days. The range of the amplitudes of vibration represented on these dials is such that average normal persons, and indeed patients with known disease involving the sensory neurons thus far tested, can perceive the vibrations practically anywhere on the surface of the body within its limits.

Determination of the Adaptation Response.—A second and more delicate evaluation of the vibratory sensibility can also be made by the pallesthesiometer, namely, the adaptation, or "fatigue," response. After the threshold value at a point had been ascertained, a heavy vibration (the vibrator operating at full 110 volts—vibration amplitude v^2 12,100—was applied to this area for a period of one minute, after which the threshold value was considerably increased. Threshold readings were then taken at intervals of thirty seconds. In the normal alert subject, the threshold returned to the original level in three minutes. If these returning values are plotted on a graph, a more or less smooth downward "adaptation curve" results.

If the patient was reporting inaccurately, widely divergent readings were obtained, indicating that the subject made his report when he thought the stimulus should have been evident rather than when it was actually felt.

For the purposes of this experiment, uniform arbitrary points were selected as follows: (1) the volar surface of the right and the left forearm, 10 cm. from the proximal edge of the ulnar styloid, in the midline; (2) the volar surface of the upper part of the right and the left arm, on the biceps muscle, 10 cm. from the medial epicondyle, in the midline; (3) the center of the right and the left patella, and (4) the anterior surface of the right and the left thigh, over the quadriceps muscle, 10 cm. from the proximal edge of the patella, in the midline.

These test points were marked in ink, so that as various readings were taken the same spot would be used each time and thus the chance for variables to enter into the results would be decreased.

Helen P. Reisman performed a portion of the technical work.

From the Research Laboratories, S. M. A. Corporation; the Department of Contagious Diseases, City Hospital, and the Department of Pediatrics, Western Reserve University.

1. (*a*) Aring, C. D., and Frohring, W. O.: Apparatus and Technique for Measurement of Vibratory Threshold and Vibratory "Adaptation" Curve, J. Lab. & Clin. Med. **28**:204-207 (Nov.) 1942. (*b*) Frohring, W. O.; Kohn, P. M., and Toomey, J. A.: The Effect of Simple Analgesics and Quinine on the Threshold of Vibratory Sensibility as Measured by the Pallesthesiometer, Am. J. Dis. Child., to be published.

PRELIMINARY TESTS

Before work was begun on patients with poliomyelitis it was decided to test a few patients with known neurologic diseases in whom no disturbance of the vibratory sense could be demonstrated by use of the ordinary tuning fork. Two of several such patients may be described in detail.

CASE 1.—A 28 year old man had a cyst of the spinal cord which exerted pressure on spinal segments T11, T12, L1, L2 and L3, resulting in weakness of both lower extremities. He revealed no abnormalities when his vibratory sense was tested with the tuning fork. He was tested with the pallesthesiometer, the vibrating disk being applied to test points on the anterior surfaces of the thighs. One minute of heavy vibration was given. The control threshold value had not been reached after eight minutes, and a typical "delayed," or prolonged high threshold, adaptation curve was obtained. The adaptation curves obtained from testing the upper extremities were perfectly normal.

CASE 2.—A 30 year old Negress had a subdural hematoma which produced hemiplegia of the left side. The adaptation curve recorded on the left thigh was a typically prolonged delayed one, whereas that of the right thigh was normal. It was not possible to detect abnormalities over the affected side with a tuning fork.

There was little point in proceeding further with studies on this type of patient, since such typical delayed curves were also obtained by Aring and Frohring[1a] from patients with various neurologic diseases, including degeneration of the posterior columns, subacute combined degeneration of the spinal cord and peripheral neuritis.

Another point which further convinced us of the accuracy of the pallesthesiometer was its ability to detect fine changes in the vibratory sense following the administration of such simple analgesics as acetylsalicylic acid.[1b]

EXPERIMENTAL PROCEDURE

Control threshold readings were taken on 25 normal subjects, who included interns, medical students and nurses. The range of dial readings for the test point located on the dorsal surface of a forearm was from 120 to 175 dial units, with an average of 145.5 dial units. This corresponds to from v^2 118.81 to v^2 612.56. The threshold values on the biceps, quadriceps and patella test points were all higher than those on a forearm. As a further check, in order to eliminate the possible effect of prolonged confinement to bed, readings were also taken on patients with various diseases other than poliomyelitis who had been in bed from two to sixteen weeks. It was found that the threshold values of these patients fell well within the normal range. The possible effect of muscle atrophy as a factor in reducing threshold values was also investigated. It was found that the threshold values of a patient recovering from severe toxic neuronitis, which left him with severely atrophied extremities, were within the normal range. Normal adaptation curves were obtained when the patient was tested over points on the involved extremities. Another patient convalescing from encephalitis associated with measles, who had moderate atrophy of the muscles due to prolonged confinement, also exhibited normal values.

Six adult patients with poliomyelitis were tested, 4 of whom had first become ill within the previous four weeks and 2 four months and three years respectively before being tested. The patients were tested by means of the pallesthesiometer for threshold values on test points on paretic limbs, as well as for adaptation curves on test points on involved extremities. A defi and marked decrease in threshold values in the involv extremities was noted, whereas corresponding readi on uninvolved extremities showed values essenti within the normal range. Thus, for 15 test points the paretic limbs, the highest threshold value recor was 126 dial units, or v^2 136.42, a reading obtained o the patella. On forearm test points the majority of readings were in the neighborhood of 100 on the di corresponding to v^2 90.63. The lowest threshold recorded was on a forearm test point, with a reading of on the dial, corresponding to v^2 62.41.

In this group of patients, nine adaptation curves w run on test points located on the surfaces of involv limbs. After a heavy vibration, 7 of the 9 patie had typical pathologic curves, characterized by sl return of the threshold values to normal. Cont adaptation curves were taken on these patients o their uninvolved limbs, and normal curves were obtain

Three of the patients were still in the hospital th to four months later, when adaptation curves a threshold values were again determined. The resu were essentially the same.

Eleven patients were subsequently tested over involv areas. Nine showed lowered thresholds; 1 showed considerably lowered threshold at one of six sites tes and 1 showed no deviation from the normal.

COMMENT

The exact meaning of the adaptation curve far from clear. It might be explained by t theory that there is some substance which "used up" in a chemical phase of medicati when a vibratory end organ is overstimulated a which in a diseased sensory unit is restored a much slower rate than normal. Such theory would harmonize with the generally ac cepted chemical phase of the visual cycle: T dark adaptation curve resulting from exposu of the eye to a bright light corresponds in a num ber of respects to the adaptation curve resulti from heavy vibratory stimulation. At any ra it can be stated with some assurance that an obviously diseased nervous structure sou change does occur that results in an abnorm adaptation response as demonstrated by t pallesthesiometer.

It should also be mentioned that, as shown this paper, in addition to the clinical demonstr tion of involvement of the sensory neurons poliomyelitis, evidence pointing to involvem of the intercalated (internuncial) neurons h also recently been reported.[2] Thus, it is becom ing increasingly clear that the term acute poli myelitis of the anterior area of the spinal co describes only one part of the pathologic pictur

SUMMARY AND CONCLUSIONS

The pallesthesiometer is able to detect min changes in vibratory sensibility that are not de

2. Kohn, P. M.; Zucker, E., and Toomey, J. A Electromyographic Studies in Poliomyelitis, to be pu lished. Kabat, H.: Personal communication to t authors.

strated by methods of testing now ordinarily d in clinical practice.

Various types of diseases affecting the nerve ues produce a characteristic type of abnormal ptation curve as recorded by the palles- siometer, characterized by a slow phase of overy following a heavy prolonged vibratory mulus.

By means of the pallesthesiometer, sensory nges which have hitherto not been demon- able in poliomyelitis despite the obvious pathologic changes in the posterior part of the cord and in sensory ganglions, have now been shown to exist. These sensory changes include a lowered threshold of vibratory sensibility, indicating a hyperesthetic type of response, and also a typical delayed type of adaptation curve.

City Hospital, Cleveland.

3395 Scranton Road, Cleveland.

Department of Contagious Diseases, City Hospital, 3395 Scranton Road, Cleveland.

Research Laboratories, S. M. A. Corporation, Chagrin Falls, Ohio.

NORMAL STANDARDS IN THE TREATMENT OF YOUNG PERSONS WITH DIABETES

JOSEPH H. BARACH, M.D.
PITTSBURGH

While in many diseases a patient cannot by any known means be restored to the normal state, it seems that maximum restoration ought to be attained by reestablishing as many normal physiologic functions as possible. At first thought one might assume that to follow the norm is simple, but the fact remains that it cannot at this time be stated what the true physiologic normal values are in the nutrition of man. The paths which lead to the desired information are beset with doubts and uncertainties, in spite of the many studies that have been made during the past thirty to forty years.

STANDARDS OF NORMAL GROWTH

A physician treating a diabetic child, realizing that he must prescribe a diet to sustain health and provide for growth in years to come, faces a real problem. The first question that presents itself concerns the nutritional state of the diabetic child as compared with an ideal normal state. Antecedent to the solution of the first question is the still more perplexing one, "What are the norms of height and weight for a child at a given age?" The physician who accepts the first set of values that meets his eye will be much happier than he who consults the works of a large number of investigators in this field. During the past year I have attempted to find acceptable values for the normal height and weight for children at various age levels, and I am convinced that it is not enough to say that a boy of 10 years will measure between 47 and 60 inches (between 119 and 152 cm.) in height and weigh between 40 and 98 pounds (between 18.1 and 44.5 Kg.), as appears from the width-weight tables of Pryor [1] and others. More explicit guides are required for the daily work of every physician. Ordinary clinical judgment dictates that a range as wide as the one mentioned will include too many children who are not normal in height and weight. Hence, such height-weight tables are not as helpful as they seem for one who is about to prescribe a diet for the diabetic child.

Construction and Use of the Growth Chart.—W... a number of height-weight tables were compared, ... siderable variation was noted. It seemed desirable ... eliminate the variations by arriving at averages ... on the largest number of measurements available. ... this purpose observations recorded by various wor... on the height and the weight of more than 400,837 ... and girls were analyzed. Age-height-weight ta... from the following sources were used:

Source	Subjects
The Child from One to Six: His Care and Training, Publication 30, United States Department of Labor, Children's Bureau, 1931, p. 17	125
Height and Weight Survey, Toronto Elementary School Children, Dominion Bureau of Statistics, Social Analysis, Ottawa, Canada, 1942	70
Faber, H. K.: A Weight Range Table for Children from 5 to 15 Years of Age, Am. J. Dis. Child. **38**:758-761 (Oct.) 1929	60
Pryor, H. B.[1]	6
Wetzel, N. C.: Physical Fitness in Terms of Physique, Development and Basal Metabolism, J. A. M. A. **116**:1187-1195 (March 22) 1941	2
Baldwin, B. T.: The Use and Abuse of Weight-Height-Age Tables as Indexes of Health and Nutrition, J. A. M. A. **82**:1-4 (Jan. 5) 1924	129
Total	400
Physical Measurement of Growth and Nutrition, American Child Health Association, New York, 1929	
Engelbach's Tables, in Holt, L. E.: Diseases of Infancy and Childhood, ed. 11, New York, D. Appleton-Century Company, Inc., 1940, pp. 17-26	
Dreyer's Standards, in Dreyer, G., and Hanson, G. F.: Assessment of Physical Fitness, New York, Paul B. Hoeber, 1921.	
Burgess Tables, in Holt, L. E.: Diseases of Infancy and Childhood, ed. 11, New York, D. Appleton-Century Company, Inc., 1940, pp. 24-25	
Gray, H., and Fraley, F.: Growth Standards: Height, Chest-Girth and Weight for Private School Boys, Am. J. Dis. Child. **32**:554-555 (Oct.) 1926	
Gray, H., and Gower, C.: Growth Standards of Height and Weight for Girls in Private Schools, Am. J. Dis. Child. **35**: 411-413 (March) 1928	

* The first six tables listed were based on 400,83... measurements; the numbers of measurements on whi... the other tables were based were not stated, but t... total must be large.

Presented before the New York Diabetes Association at the New York Academy of Medicine, Feb. 14, 1944.

From the School of Medicine and the Renziehausen Foundation for the Study and Treatment of Diabetic Children, University of Pittsburgh.

1. Pryor, H. B.: Width-Weight Tables, Stanford University, Calif., Stanford University Press, 1940.

rst the means of the values for height and eight at each age were determined for each . Then the average of these means was lated and the resulting value used in con- ting the accompanying graphs (chart 1). solid lines represent normal growth over ies of years. The broken lines above and w the solid line represent a 10 per cent varia- above and below the theoretic ideal normal. e height and the weight of a growing child recorded from year to year over the normal hs on these charts, variations from the nor- trends appear in striking contrast.

epresentative Individual Charts. — Four ts (chart 2, *A, B, C, D*) are presented here llustrate the appearance of abnormal varia- s. Chart 2 *A* is that of a boy who is short stocky; at the age of 18 he is normal in ht but 3 inches (7.5 cm.) below normal in ht. Chart 2 *B* is that of a boy at age 14 who full normal weight but 5 inches (12.5 cm.) w normal in height. Chart 2 *C* depicts the wth of a boy who was strikingly above the age at age 11 and who grew little during next four years. Chart 2 *D* shows the growth e of a girl who was below normal in weight ten years and then passed the average in ht but never attained normal height. These ts tell the story of a child's growth at a ce. They show the progress in height and ht simultaneously, and they reveal devia- s from normal trends at the time of their rrence. As a part of the patient's record, ewed at regular intervals, they warn the sician in plenty of time when it is advisable exercise control of the diet.

NORMS IN NUTRITION

When the physician has determined the ap- ximate normal height and weight for a child given age, the question arises whether such mal levels can be attained and if so how they to be attained. On one point most physi- ns agree: the growth of a child can be in- nced only within the range of the innate acity of that child to grow, and capacity is imited by inheritance. Nevertheless, it is e that within the range determined by in- itance, obesity is created in some children by essive feeding, and undernutrition is induced others by underfeeding.

There are times when endocrine therapy stim- tes growth and seems to correct certain de- encies, but in medical experience to date, one st still admit many failures.

Granting the validity of these limitations, I w come to the gist of the problem. If one is to plan for the nutritional restoration of a diabetic child and for a normal nutritional level throughout the remainder of the period of growth, what shall be the approach? What is normal, and what is optimal?

Caloric Allowance per Day.—Table 1 shows the lack of unanimity among authorities as to the optimal daily caloric requirement for normal children. The summary of data in the last horizontal column shows that the range of variation is wide. One might prescribe from 20 to 300 per cent more or fewer calories for a child of a certain age, depending on which of these standards one happened to accept. It is evident that whichever standard one adopts, a recognized authority for one's choice can be found.

Protein Allowance per Day.—Proteins in the normal diet: The recommendations of various authors as to the daily allowance of protein vary as much as 200 per cent (table 2). Contributors to the report of the League of Nations recommended from 2.0 to 3.5 Gm. of protein per kilogram of body weight per day, while other authorities allowed up to 4 Gm. of protein per kilogram for children 1 to 3 years of age.[1a] Can it be that 2, 3 and 4 Gm. of protein per kilogram are equally good physiologically and that it does not matter at all whether a child at a certain age is given 18 Gm. or 40 Gm. of protein per day? If it does not matter, the assumption that physicians are following a known scientific procedure seems illogical, since actually there may be a variation of 100 per cent or more.

The need for an adequate amount of protein in the daily diet in health and in disease has come to be fully appreciated. Of late more attention is being paid than ever before to hypoproteinemia, and diets relatively high in protein are being prescribed for its relief, but it must be remembered that increased feeding of protein does not necessarily produce an increase of protein in the plasma. The relationship between these two values is far from constant: The amount of protein in the plasma depends on the physiologic integrity of the liver more than it does on the amount of protein ingested from day to day.

On the other hand, recent studies reported by Stare and Thorn [2] and others indicate that an active adult, a soldier in training, will maintain good health on 50 Gm. of protein per day, even if only 5 Gm. of that amount is in the form

1a. Lewis, H. B.: Proteins in Nutrition, in Handbook of Nutrition, American Medical Association, 1943, p. 30.

2. Stare, J. F., and Thorn, G. W.: Some Medical Aspects of Protein Foods, Am. J. Pub. Health **33**:1444-1450 (Dec.) 1943.

of animal protein. These authors stated the belief, which they shared with Chittenden,[8] that exercise and manual labor do not materially increase the protein requirement; and yet, relatively large amounts of protein are given growing children, as much for meeting the quirements of their physical activity as to p vide for growth.

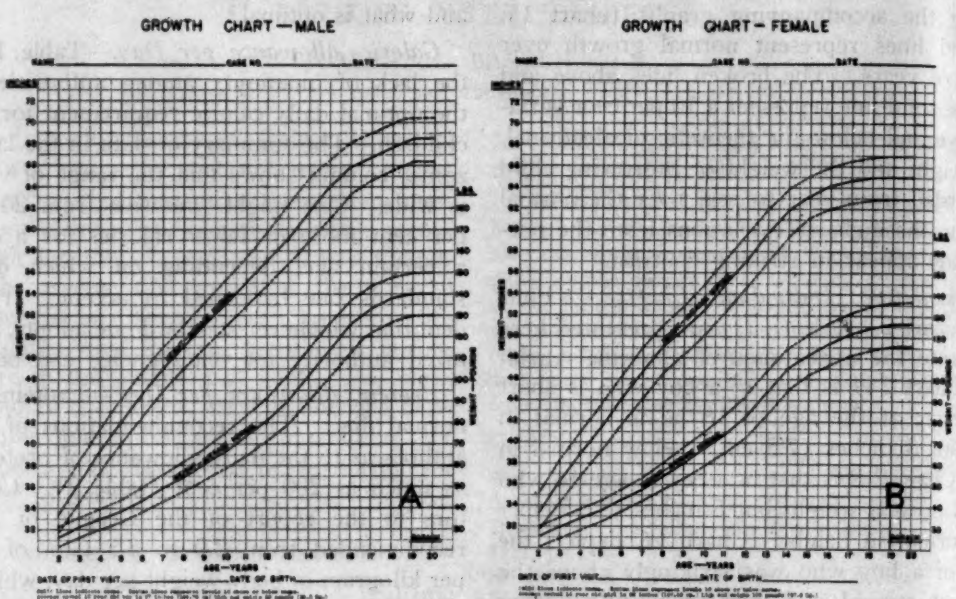


Chart 1.—Age-height-weight chart for (*A*) boys, (*B*) girls.

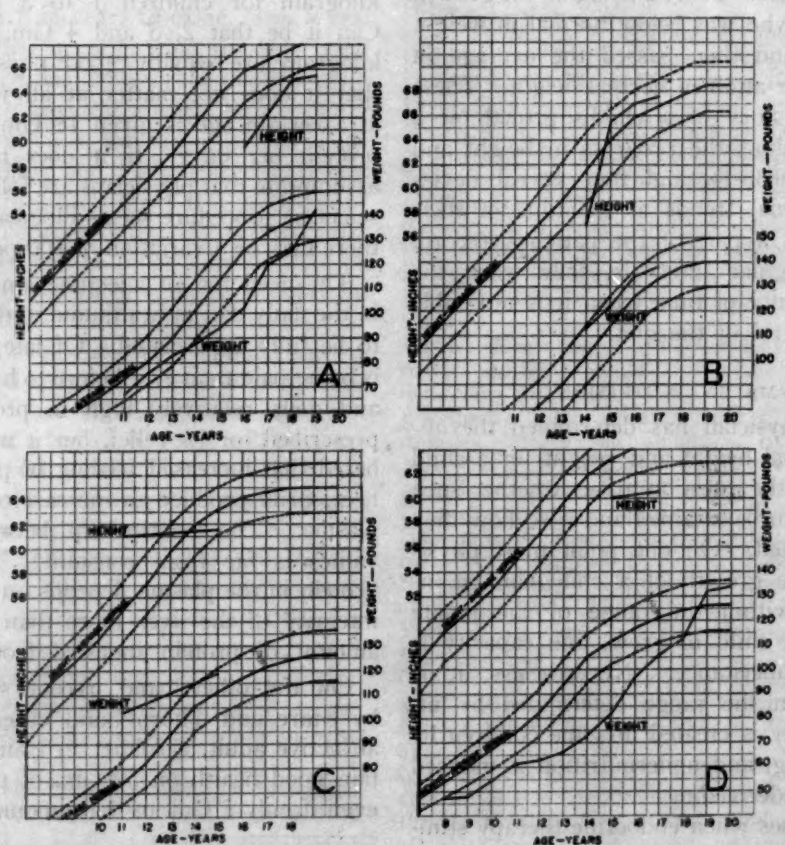


Chart 2.—Representative charts: *A*, a short, stocky boy; *B*, a boy of normal weight but of subnormal heig *C*, a boy whose growth was inadequate for four years; *D*, a girl of normal weight and subnormal height.

3. Chittenden, R. H.: Physiological Economy in Nutrition, Philadelphia, F. A. Stokes Co., 1913, p. 127.

Other studies have been made on the phys logic adequacy of diets relatively low in prote

Source	1 Yr.	2 Yr.	3 Yr.	4 Yr.	5 Yr.	6 Yr.	7 Yr.	8 Yr.	9 Yr.	10 Yr.	11 Yr.	12 Yr.	13 Yr.	14 Yr.	15 Yr.	16 Yr.	17 Yr.	18 Yr.	19-20 Yr.
Medical Research Council, quoted by Drummond, J. C.: Lane Medical Lectures: Biochemical Studies of Nutritional Problems, Stanford University, Calif., Stanford University Press, 1934, p. 23	.....	.....	.....	.....	.....	820 to 2,460	900 to 2,700	970 to 2,910	1,020 to 3,060	1,060 to 3,180	1,120 to 3,360	1,200 to 3,600	1,250 to 3,750	1,310 to 3,930	1,350 to 4,050	Energy requirement 3 × metabolic rate			
New Health Society Report, London, 1927, quoted by Drummond, J. C.: ibid., p. 23	Public school		.....	.....	.....	.....	.....	.....	.....	3,350	3,350	3,300	3,300	3,300	3,300	3,300	3,300		
	Boys school		.....	.....	.....	.....	.....	.....	3,146	3,146	3,146	3,500 3,146	3,500 3,146	3,500 3,146	3,500 4,129	3,500 3,146	3,500 4,129		
DuBois, E. F., and Chambers, W. H.: Calories in Medical Practice, in Handbook of Nutrition, Chicago, American Medical Association, 1943, p. 55	.....	.....	1,200	1,600	1,600	1,600	2,000	2,000	2,000	2,500	2,500	2,500	2,800	2,800	2,800	♂ 3,800 ♀ 2,400	♂ 3,800 ♀ 2,400	♂ 3,800 ♀ 2,400	♂ 3,800 ♀ 2,400
National Research Council, quoted in Recommended Allowances for the Various Dietary Essentials, Current Comment, J. Am. Dietet. A. **17**: 565-567 (June-July) 1941	1,200	1,200	1,200	1,600	1,600	1,600	2,000	2,000	2,000	2,500	2,500	2,500	♂ 3,200 ♀ 2,800	♂ 3,200 ♀ 2,800	♂ 3,200 ♀ 2,800	♂ 3,800 ♀ 2,400	♂ 3,800 ♀ 2,400	♂ 3,800 ♀ 2,400	♂ 3,800 ♀ 2,400
The Problem of Nutrition: Report on the Physiological Bases of Nutrition, League of Nations Publication, New York, World Peace Foundation, 1936, vol. 2, pp. 13-14	.....	840	1,000	1,200	1,200	1,440	1,440	1,680	1,680	1,920	1,920	2,160	2,400	2,400	2,400	2,400	2,400	2,400	2,400
											For muscular work add approximately 600 calories								
White, P.: Diabetes in Youth, New England J. Med. **224**: 586-589 (April 3) 1941	1,000	1,100	1,200	1,300	1,400	1,500	1,600	1,700	1,800	1,900	2,000	2,100	2,200	2,300	2,400	2,500	2,600	2,700	2,800
Range within various standards.....From	1,000 to 1,200	840 to 1,200	1,000 to 1,200	1,200 to 1,600	1,200 to 1,600	820 to 2,460	900 to 2,700	970 to 2,910	1,020 to 3,146	1,060 to 3,350	1,120 to 3,360	1,200 to 3,600	1,250 to 3,750	1,310 to 3,930	1,350 to 4,129	2,400 to 3,800	2,400 to 4,129	2,400 to 3,800	2,400 to 3,800

TABLE 2.—*Daily Protein Allowance Recommended by Various Authorities for Normal Children (Grams per Kilogram of Body Weight)*

Source	1 Yr.	2 Yr.	3 Yr.	4 Yr.	5 Yr.	6 Yr.	7 Yr.	8 Yr.	9 Yr.	10 Yr.	11 Yr.	12 Yr.	13 Yr.	14 Yr.	15 Yr.	16 Yr.	17 Yr.	18 Yr.	21 Yr.
Nobécourt, in The Problem of Nutrition: Statistics of Food Production, Consumption and Prices, League of Nations Publication, New York, World Peace Foundation, 1936, vol. 4	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	...	...	...	...	...	2.0	...	...	...	...	...
Lesné, E.: Nutrition in Childhood, Quart. Bull. Health Organ., League of Nations **5**: 531, 1936; the reference is to page 534	2.75	2.75	2.75	2.75	2.75	2.75	2.75	2.75	2.75	2.75	2.75	2.75	2.75	...	...	2.0	...	1.5	...
Kugelmass, I. N.: The Newer Nutrition in Pediatric Practice, Philadelphia, J. B. Lippincott Company, 1940	...	3.0	3.0	3.0	3.0	2.5	2.5	2.5	2.5	2.5	2.5	2.5	...	...	...	...	...	...	...
Richet, C., in The Problem of Nutrition: Statistics of Food Production, Consumption and Prices, League of Nations Publication, New York, World Peace Foundation, 1936, vol. 4	3.5	3.5	3.0	3.0	3.0	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.0	2.0	1.5	1.5	1.0
The Problem of Nutrition Statistics of Food Production, Consumption and Prices, League of Nations Publication, New York, World Peace Foundation, 1936, vol. 2, pp. 13-14.	3.5	3.5	3.0	3.0	3.0	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.0	...	1.5	...	1.0
Lewis, H. B.: Proteins in Nutrition, in Handbook of Nutrition, Chicago, American Medical Association, 1943, p. 30	4.0	4.0	4.0	...	...	...	...	...	...	...	...	...	...	...	...	...	2.0	2.0	...
Food Charts Prepared by a Joint Committee of the Council on Foods and Nutrition of the American Medical Association and of the Food and Nutrition Board of the National Research Council, Chicago, American Medical Association, 1942, p. 18 *	40	40	40	50	50	50	60	60	60	70	70	70	85♂ 80♀	85♂ 80♀	85♂ 80♀	100 75	100 75	100 75	100 75

* Allowance expressed as total number of grams per day.

Strieck [4] reported metabolic studies on adults who maintained nitrogen equilibrium with an intake of 23 to 40 Gm. of protein per day while engaging, over many years, in arduous physical effort, such as repeated mountain climbing to the level of 14,000 feet (462 kilometers).

Protein in the Diet of Foreign Peoples: Observations on the dietary habits of various peoples have been made from time to time. One that is of interest with respect to the relationship between physical exertion and intake of animal protein is the observation reported by Hugo Byas [5] on figures published by the Japanese. According to Byas, the consumption of meat in Japan was 4 pounds (1.8 Kg.) per person per year, while in the United States the consumption of meat at the same time was approximately 145 pounds (66 Kg.) per person per year. Still more striking is the fact that the American soldier of today (1942 to 1944) is being given 280 pounds (127 Kg.) of meat per year.

The recent report by Shen [6] on the diet of Chinese soldiers and students is also interesting. Shen reported that the daily ration of a group of 11,388 Chinese soldiers consisted of rice, leafy vegetables, a few tubers and a small amount of added fat. These rations contained 762 Gm. of carbohydrate, 76 Gm. of vegetable protein and 10 Gm. of fat per day. A group of 160 Chinese university students subsisted on 342 Gm. of carbohydrate, 41 Gm. of protein and 20 to 30 Gm. of fat per day.

These statements of fact pertaining to protein requirement reveal a striking lack of agreement on what is physiologic or normal, and they give added import to the statement recently voiced by Carlson [7] that "the sciences of physiology, nutrition and medicine do not today have sufficient knowledge to outline the components of an optimum diet."

Protein in the Diet of the Diabetic Patient: What has been said of the protein requirement up to this point pertains to the diet for persons in health. When one considers the allowance of protein for a diabetic patient, other factors enter into consideration. In diet high in protein the amount of nitrogenous waste is increased, and the patient who is to take good care of increased nitrogen should have an adequate water exchange. This the patient with diabetes does not have, as is evidenced by the polyuria and polydipsia, the desiccation of tissue with loss of weight and the azotemia which are present in diabetic ketosis and coma.

The liver of the diabetic patient is a mill of pathologic physiology, and it is highly involved in the metabolism of protein. To overload the liver of a diabetic patient with a large protein intake than is necessary can lead only to the accumulation of more waste and to further destruction. Everything that is known today concerning the function of the liver in diabetes points to the inadvisability of a diet unnecessarily high in protein.

Carbohydrate and Fat Portions in the Diet of the Diabetic Patient.—The diet which I consider adequate for normal adults obtains approximately 65 per cent of its calories from carbohydrate, 15 per cent from protein and 20 per cent from fat. These values correspond closely to those of the Nutrition Committee of the League of Nations, as well as figures of Voit. In planning diets for adult diabetic patients since the advent of insulin, I have tried to keep as closely as possible to these normal standards.[8] For persons with severe, the so-called total, diabetes for whom large portions of carbohydrate would require the use of unusually large amounts of insulin, the portion of fat is increased to a limited degree. At no time, however, is the caloric value of the fat portion of my diets allowed to exceed the caloric value of the combined carbohydrate and protein.

The diet for children with diabetes differs from that for adults. Children require more milk, and this with the usual amount of butter introduces a larger amount of fat. It should be noted here that human milk, the complete food of early life, obtains 40 calories from combined carbohydrate and protein and 31.5 calories from fat. Cow's milk obtains 46 calories from carbohydrate and protein and 31.5 calories from fat. In both instances more calories are derived from carbohydrate and protein than from fat.[9]

Adequate Diets for Diabetic Children—a Table of Diets.—A table of diets for diabetic children (table 3) was constructed on the basis of (1) normal body weights in successive years (2) adequate caloric portions, (3) adequate protein portions, (4) adequate carbohydrate portions and (5) adequate fat portions. Each of these values approximates the normal level as nearly as possible

4. Strieck, F.: Metabolic Studies in a Man Who Lived for Years on a Minimum Protein Diet, Ann. Int. Med. **11**:643-650 (Oct.) 1937.

5. Byas, H.: Behind Japan Boom in Trade, New York Times Mag., May 26, 1935, sect. 7, p. 2.

6. Shen, T.: Diet of Chinese Soldiers and College Students in Wartime, Science **98**:302-303 (Oct. 1) 1943.

7. Carlson, A. J.: Some Obstacles in Path Towards Optimum Diet, Science **97**:385-390 (April 30) 1943.

8. Barach, J. H.: Normal Standards in the Treatment of One Hundred Cases of Diabetes with Insulin, J. A. M. A. **82**:347-351 (Feb. 2) 1924.

9. Jeans, P. C.: Feeding of Healthy Infants and Children, J. A. M. A. **120**:913-921 (Nov. 21) 1942.

From this table a maintenance diet appropriate the age of the patient is prescribed at the inning of treatment and is continued during current year as long as the patient makes isfactory and normal progress. If and when re food is indicated in order to maintain rmal growth and health, the quantities are reased.

ROWTH OF 135 CHILDREN WITH DIABETES IN RELATION TO THEIR DIET

A group of 135 children were under diabetic trol for two to twelve years, during which e their age, height and weight were recorded frequent intervals. For the purpose of this dy a graph was made for each of the 135 ldren, and this graph was superimposed on the normal weight, and 15 were more than 10 per cent above normal. Table 4 summarizes these findings.

The caloric value of the diets on which these children had been subsisting was compared with the caloric values of the diets for the respective ages shown in table 3. If the caloric value of a child's diet was below that of the minimum diabetic diet (the lowest caloric value I now prescribe), that child was classified as having been on an inadequate diet. If the caloric value was as high as that of the minimum diet, the child was classified as having been on an adequate diet. If the caloric value was as high as that of the maximum diet, which is the highest value that I now prescribe, the child was also classified as having been on a normal diet. If the caloric value was in excess of the maximum diabetic diet, the patient was classified as having been on an above maximum diet.

TABLE 3.—*Maintenance Diet for Diabetic Children* *

ge, ars	Weight		Maximum Diet, Daily Allowance				Minimum Diet, Daily Allowance			
	Pounds	Kg.	Calories	Carbohydrate, Gm.	Protein, Gm.	Fat, Gm.	Calories	Carbohydrate, Gm.	Protein, Gm.	Fat, Gm.
½	15	7	700	90	30	25	600	75	25	25
1	20	9	1,000	125	40	35	800	100	30	30
2	26	10	1,100	140	45	40	880	110	35	35
3	31	14	1,300	150	50	45	960	120	40	40
4	35	16	1,300	165	50	50	1,040	130	40	40
5	38	17	1,400	175	55	55	1,120	140	45	45
6	43	19	1,500	185	55	60	1,200	150	46	50
7	50	22	1,600	200	65	60	1,280	160	50	50
8	55	25	1,700	215	65	65	1,360	170	55	55
9	61	28	1,800	225	70	70	1,440	180	55	55
10	67	30	1,900	235	70	75	1,520	190	55	60
11	75	34	2,000	250	70	80	1,600	200	65	65
12	81	37	2,100	265	80	80	1,680	210	65	65
13	90	40	2,200	275	85	85	1,760	220	70	70
14	103	47	2,300	285	85	90	1,840	230	70	70
15	112	50	2,400	300	90	95	1,920	240	75	75
16	126	57	2,500	315	95	95	2,000	240	80	80
17	133	60	2,600	325	100	100	2,080	240	80	90
18	138	63	2,700	340	100	105	2,160	240	85	95
19	138	63	2,800	350	100	110	2,240	240	90	100
20	139	63	2,900	365	100	115	2,320	240	95	110

* In this table fractions have been disregarded in favor of round numbers.
Caloric values of minimum diets are 20% below those of maximum diets; they may be increased to full maximum when irable
These diets obtain 50% of their calories from carbohydrate, 15% from protein and 35% from fat.
After the age of 15, when the growth rate slows, carbohydrate portions remain the same; fat may be increased. A diet er in fat may be attained by substituting 2 Gm. of carbohydrate for 1 Gm. of fat in the daily diet.
After the age of 16, girls weigh 10% less than boys.

e growth chart. If the height and weight lines a child approximated the normal, that child as classified as normal; if the graph was above below the normal levels the child was classied as oversized or undersized. Of the 135 chilen, 25 proved to be normal. Of these 25 patients, all were less than 2 inches (5 cm.) above below the ideal height and less than 5 pounds 2.3 Kg.) above or below the ideal weight.

Fifty-seven children were classed as undersized. Of these, 51 were below the normal ight and 31 were more than 10 per cent of e total height below normal. Fifty-four of e 57 were below the normal weight, and 31 ere more than 10 per cent below normal.

Fifty-three children were classed as oversized. f these, 50 were above the normal height and were more than 10 per cent above the normal eight. Forty-six of the 53 children were above

TABLE 4.—*Growth of 135 Children with Diabetes*

		Height		Weight	
Growth	Number of Patients	Above or Below Normal	More Than 10% Above or Below Normal	Above or Below Normal	10% Above or Below Normal
Normal	25	25 Slight variation	0	25	0
Undersized	57	Below 51	Below 31	Below 54	Below 31
Oversized	53	Above 50	Above 39	Above 46	Above 15

As will be seen from table 5, 9 children attained normal growth on diets of lower caloric value than the minimum diet, and 16 did not exceed normal growth although their diets were excessive. In the first instance there may have

been a sparing of the metabolism, or it may be that the minimum diet was altogether adequate for these 9 children. The fact that 16 children had excessive diets but did not grow beyond the normal apparently means that other limiting factors were at work.

Of the 57 undersized children, 30 had less than the minimal requirement shown in the table, and 27 had the full minimal requirement. None had the maximum diet or more than the maximum diet.

Of the remaining 53 children, all were oversized. Thirty-one received the maximum diet, and 22 had more than the maximum diet.

Taken as a whole these data reveal that some children attained normal growth on diets of lesser caloric value than I ordinarily prescribe and that others did not exceed normal growth, regardless of the fact that they had been given diets of greater value than I consider necessary for normal growth. The data also reveal that the undersized children had been living on inadequate diets, or, at most, on diets that I consider minimal for normal growth. The oversized children had been having as much as my maximum allowance calls for, and in 22 of 53 cases more than my maximum diet.

TABLE 5.—*Growth Attainment of 135 Children with Diabetes in Relation to Diet*

Growth	Number of Patients	Inadequate Diets	Adequate Diets	Maximum Diets	Above Maximum Diets
Normal	25	9	..	..	16
Undersized	57	30	27	..	..
Oversized	53	0	0	31	22

These studies reveal a positive relationship between the caloric value of the diet and the growth of the child. All overfed children may not attain oversize, nor will all conservatively fed children remain undersized, but in the majority of children in this series whose diets were too conservative or too generous, undersize or oversize occurred.

INSULIN REQUIREMENT AND DIETS PRESCRIBED FOR 125 CHILDREN WITH DIABETES

In order that the relationship between diet and insulin requirement might be studied 125 patients were divided into two groups, conservatively fed and generously fed children. There were 41 patients in the first group and 84 in the second. These groups were further subdivided into children of normal growth, undersized children and oversized children. It will be noted from table 6 that the amount of insulin required by normal and by undersized children on conservative diets was about the same. This was also true for the generously fed children although the generously fed required considerably more insulin. The oversized children who were receiving a conservative diet required a slightly larger amount and those who were receiving a generous diet required a still larger amount of insulin than either the normal or the undersized children. For the series as a whole it will be noted that while the average

TABLE 6.—*Insulin Requirement and Type of Diet*

Growth	Conservative Diet: Number of Cases	Conservative Diet: Insulin, Average Dose	Generous Diet: Number of Patients	Generous Diet: Insulin, Average Dose
Normal	8	29	15	44
Undersize	20	32	35	48
Oversize	13	36	34	68
Total	41	32	84	55

amount of insulin for the conservatively fed children was 32 units per day, the generously fed children required an average of 55 units per day, nearly twice as much.

SUMMARY

After a review of present day standards of age-height-weight values, charts based on averages obtained from a large number of measurements were constructed. These charts portray the normal trends of growth from the ages of 1 to 21 years. On the basis of the generally accepted values for caloric requirement per day and daily protein requirement per kilogram of body weight by growing children and of the amounts considered normal and adequate for children of various ages, a table of diets was constructed to serve as a standard for this study. The growth curves of 135 children with diabetes who had been under observation for two to twelve years were superimposed on the normal growth charts, and this revealed that some of the children were of normal size, others were below normal and the remainder were oversize. The caloric values of the diets on which these children had been subsisting were compared with those of normal or adequate diets, and it was found that some children attained normal growth on diets of lesser caloric value than I ordinarily prescribe and others did not exceed normal growth, regardless of the fact that they had been given diets of greater value than I consider necessary for normal growth. The diet of the oversized children was the equal of the maximum or the maximum diet (table 5).

The generously fed children required from 1.5 to nearly 2 times as much insulin as those who had been subsisting on conservative diets.

3601 Fifth Avenue.

Case Reports

ERYTHROBLASTOSIS FETALIS IN MOTHERS WITH RH-POSITIVE BLOOD

REPORT OF SIX CASES, WITH COMMENT ON ISOIMMUNIZATION WITH THE "A" AND "B" AGGLUTINOGENS

S. H. POLAYES, PH.B., M.D.

BROOKLYN

The recent work of Dr. Philip Levine and his workers [1] has established two fundamental cepts: (1) the induction of isoimmunization a pregnant woman by antigens of the fetus products of conception), inherited from the er, and (2) pathogenesis of the various ternal and neonatal diseases which may result m isoimmunization. Dr. Levine's work has olutionized some of the teachings of immunol-sts, and therefore it is fitting that these new cepts be named the Levine phenomenon.

The theory of isoimmunization was developed Levine and Stetson [2] in 1937, from the study a patient who, after delivery of a child, had ransfusion reaction to blood of her own group. r blood, as well as that of her donor, belonged group O; yet her serum was found to con-n atypical agglutinations which clumped the cells of most group O bloods. This atypical glutinin disappeared after several months, a t which suggested to the authors that the glutinin had been induced by an antigen pres- in the fetus; therefore, the atypical aggluins were considered immune bodies. The igen which induced the isoimmunization was bsequently identified as the Rh factor, discov-d by Landsteiner and Wiener [3] about two rs later.

Since then, Levine and his co-workers have proved beyond doubt that in over 90 per cent of cases the pathogenesis of erythroblastosis fetalis is based on isoimmunization of the mother with Rh-negative blood by the Rh factor present in the blood of the fetus. However, antigens other than Rh are capable of inducing isoimmunization in a similar manner. This fact was demonstrated in a case reported by Levine and Polayes [4] in 1941. In this instance, as in the first case of Rh isoimmunization reported by Levine and Stetson, the postpartum transfusion reaction was found to be due to an atypical antibody in the patient's serum, presumably resulting from previous isoimmunization of the patient by her pregnancy. However, this immune body was an atypical hemolysin instead of an anti-Rh agglutinin, the patient having Rh-positive blood. This hemolysin, like the anti-Rh agglutinin, gradually diminished in titer, and therefore was also considered an antibody induced by isoimmunization during pregnancy. Later, in a case of erythroblastosis reported by Levine,[5] Javert and Katzin, isoimmunization was again shown to have been produced by an antigen other than Rh, this time by one which in Levine's opinion is genetically related to the Rh factor and which he has termed Hr. It will be noted that in both of these cases, significantly, the patient's blood was Rh positive.

Erythroblastosis fetalis, therefore, may result from isoimmunization induced by antigens other than Rh. This throws considerable light on some of the old observations regarding the relationship of heterospecific pregnancies to maternal and neonatal diseases. Thus, Ottenberg,[6] as far back

Read before the Brooklyn Academy of Pediatrics, ril 26, 1944.

From the Department of Pathology, Cumberland and ospect Heights Hospitals.

1. Strauss, T. W.: The Rh Factor, Roche Rev. 267, 1944. This paper includes the most complete erence list on this subject to date, with a chronoic summary of the development of knowledge conming the Rh factor and erythroblastosis. It contains references listed alphabetically, including 21 of Philip Levine's more important publications on this bject.

2. Levine, P., and Stetson, R. E.: An Unusual pe of Intragroup Agglutination, J. A. M. A. **113**: (July 8) 1939.

3. Landsteiner, K., and Wiener, A. S.: An Agglutable Factor in Human Blood Recognized by Immune ra for Rhesus Blood, Proc. Soc. Exper. Biol. & Med. :223, 1940.

4. Levine, P., and Polayes, S. H.: An Atypical Hemolysin in Pregnancy, Ann. Int. Med. **14**:1903, 1941.

5. Levine, P.: Mechanism of the Isoimmunization by the Rh Factor of Red Blood Cells, Arch. Path. **37**: 83 (Jan.) 1944.

6. Ottenberg, R.: Etiology of Eclampsia: Historical and Critical Notes, J. A. M. A. **81**:295 (July 28) 1923.

as 1923, suggested the possibility of interaction of the A and B agglutinogens between mother and fetus with resultant pathologic processes. In 1928, Polayes and associates [7] concluded from a study of cord, fetal and maternal bloods in 1,000 cases that an interchange of antigens and antibodies probably does occur between mother and fetus through the placenta but that by some "protective mechanism" pathologic effects are prevented. In 1936, Jonsson [8] demonstrated the antigenic effect of the A and B agglutinogens in human beings, as did others later.

In the light of present knowledge it appears that the "protective mechanism" just mentioned is based on the fact that the A and B antigens (or group-specific substances) are present not only in the red cells but in the tissue (somatic) cells and the body fluids, where the A and B antigens can bind their specific anti-A or anti-B agglutinins, thereby inhibiting them and preventing them from producing any pathologic effects. Eighty per cent of the population—the so-called secretors—are protected by this mechanism. In the case of the Rh antigen, however, this "protective mechanism" cannot be employed by the body, since the Rh factor (so far as is known from studies made up to the present time) is present only in the red cells. This may explain the frequency of pathologic results (erythroblastosis fetalis, premature births, stillbirths, miscarriages, toxemias, etc.) in cases of Rh isoimmunization as compared with cases of isoimmunization by antigens such as A and B.

However, the workings of the "protective mechanism" against the group-specific substances just referred to are not always entirely successful, and as a result an occasional pathologic effect is encountered. This result seems to be based on isoimmunization with the A and B agglutinogen, as, for example, in instances of erythroblastosis fetalis occurring when the mother's blood is Rh positive and the offspring's blood is incompatible in heterospecific pregnancy. At this point it may be stated that soon after Levine revealed the mechanism of isoimmunization and the role which the Rh factor plays in the pathogenesis of erythroblastosis, it became apparent that the A and B antigens could induce isoimmunization with all its various pathologic effects in a manner similar to that of the Rh factors. Of course, as was pointed out by Dr. Levine at that time as well as later,[9] this statement applies only to cases in which the infants are members of the 20 per cent nonsecretors (persons in whom the A and B antigens, like the Rh factor, appear to be limited to the red blood cells).

With the more widespread testing of patients for the Rh factor, especially mothers in whose offspring erythroblastosis is anticipated, more and more of these instances are beginning to appear. Two of 6 similar cases of this type are reported in detail here. (The histories of the other 4 are not sufficiently complete for publication.) These cases were encountered by the author among a personal collection of about 1[illegible] cases of erythroblastosis fetalis, a large proportion of the data on which has already been published by Dr. Levine.[1]

REPORT OF CASES

CASE 1.—A boy delivered by Dr. Charles Weitzman on Aug. 13, 1943 was observed about six hours after birth to be jaundiced. (Since the child was born at night, a slight jaundice at birth may have been overlooked.) The child was born at term by a normal spontaneous delivery; he weighed 6 pounds (2,722 Gm.) at birth.

This baby represented a second pregnancy, the first child having died of congenital heart disease at 3 months of age. Both pregnancies were normal throughout their course.

Examination of the infant soon after jaundice was noted showed little if any enlargement of the liver or the spleen. The blood smear, however, showed 10 per cent normoblasts and numerous immature white cells. The jaundice progressed, and the child became increasingly responsive. On Aug. 15, 1943, two days after jaundice was first noted, there were still 10 per cent normoblasts present. A transfusion of 125 cc. of group O Rh-negative blood was given and was followed by definite improvement. (Rh-negative blood was used because a complete examination of the mother's blood had not yet been made.) This procedure was repeated four days later; the jaundice then subsided, and the child made a rapid recovery. He was discharged and cured on Aug. 28, 1943, at 15 days of age, with the diagnosis of erythroblastosis fetalis (icterus gravis type).

Examination of the mother's blood showed that the red cells were group O, Rh positive. The serum showed an anti-A agglutinin titer of 1:750 and an anti-B agglutinin titer of 1:250. Examination of the baby's blood showed the red cells to be group A, Rh positive.

CASE 2.—A boy was delivered spontaneously at Cumberland Hospital, Brooklyn, after nearly eight and one-half months of gestation. The child weighed 4 pounds (1,814 Gm.) and presented slight jaundice at birth.

7. Polayes, S. H.; Lederer, M., and Wiener, A. S.: Studies in Isohemagglutination: II. The Landsteiner Blood Groups in Mothers and Infants, J. Immunol. **17**: 545, 1929.

8. Jonsson, B.: Zur Frage der heterospezifischen Schwangerschaft, Acta path. et microbiol. Scandinav. **13**: 424, 1936.

9. (*a*) Levine, P.: Personal communication to the author. (*b*) Levine, P.; Burnham, L.; Katzin, E. M. and Vogel, P.: The Role of Isoimmunization in Pathogenesis of Erythroblastosis Fetalis, Am. J. Obst. & Gynec. **42**:925, 1941. (*c*) Levine, P.: Erythroblastosis Fetalis and Other Manifestations of Isoimmunization, West. J. Surg. **50**:468, 1942; (*d*) Serologic Factors as Possible Causes in Spontaneous Abortion, J. Hered. **34**:71, 1943.

he child represented the mother's fifth pregnancy. r first child is at the time of writing 11 years old well. The second child is 7 years old and well was jaundiced at birth. The third pregnancy ended a miscarriage after six months. The fourth pregcy ended in a still birth at term.

The patient appeared to be well except for progressive ndice. The blood count gave these results: red od cells, 3,250,000 per cubic millimeter, with marked ychromasia and 5 per cent normoblasts; hemoglobin, 2 Gm. per hundred cubic centimeters; white blood s, 8,000 per cubic millimeter, with 49 per cent trophils, 47 per cent lymphocytes and 4 per cent nocytes. The coagulation time and the prothrombin e were normal.

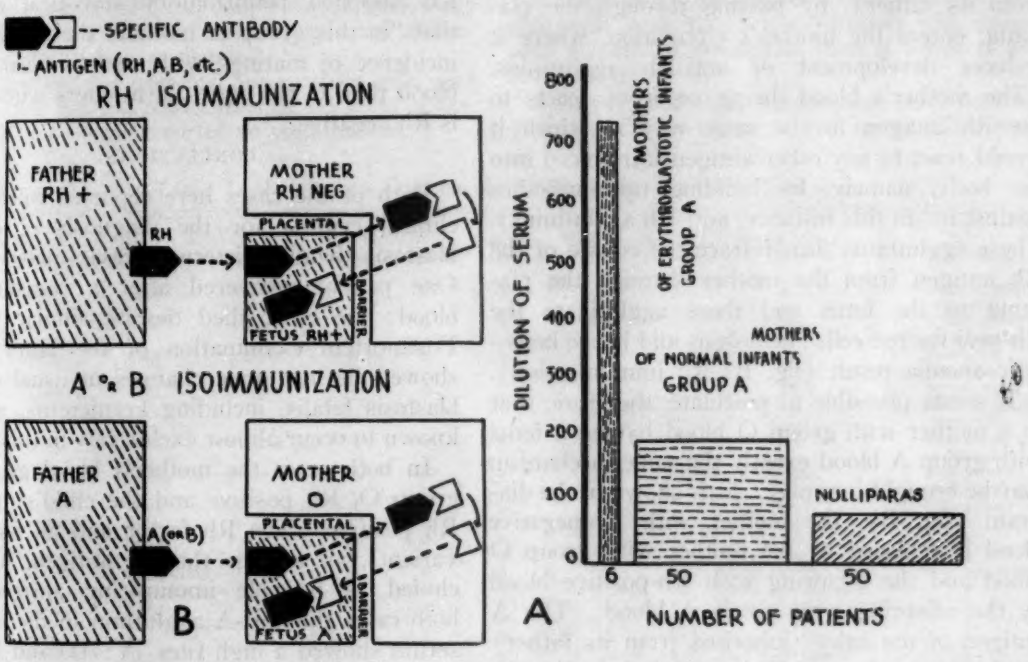
The patient began to decline about twenty-four hours r birth, and two transfusions of group A blood re administered. Despite the therapy, the jaundice ame progressively worse, petechiae, epistaxis and spiratory distress developed and the patient died at days of age.

The postmortem examination disclosed: (1) generald icterus with marked kernicterus; (2) anemia, tramedullary hemopoiesis and multiple visceral hemorages, and (3) prematurity (eight and one-half nths).

Examination of the mother's blood showed that the d cells were group O, Rh positive. The serum showed anti-A agglutinin titer of 1:700 and an anti-B glutinin titer of 1:250. Examination of the baby's od showed the red cells to be group A, Rh positive.

COMMENT

Both of these babies had mothers with Rh-sitive blood in whom the possibility of isomunization by any of the variants of the h factor as well as by Hr was excluded. Dr. Philip Levine checked all tests before reults were considered acceptable.) For both fants the diagnosis of erythroblastosis fetalis seemed fairly well established clinically, and for the infant who came to necropsy it appeared to be established anatomically as well. In the case of the latter patient the presence of kernicterus may be considered almost pathognomonic, since this lesion, as far as I could determine, occurs exclusively in patients with erythroblastosis fetalis.[10] In each case, including the 4 cases not reported in detail here, the mother's blood was of group O and the child's of group A, and in each case the anti-A agglutinin in the mother's serum reached the comparatively high titer of 1:700 or over. Unfortunately, it was not possible to determine whether the titer had declined since the first examination of the serums, but there is considerable presumptive evidence that the high anti-A agglutinin titer observed in these mothers was due to isoimmunization induced by the A antigen of the fetus. The increased titer of the anti-B agglutinin observed in both cases, while not entirely understood, is probably a nonspecific response such as is observed not infrequently in immunologic reactions.



A, serum anti-A agglutinin titration of group O women; *B*, diagram of mechanism of isoimmunization.

For purposes of comparison, the bloods of approximately 50 consecutive women with group O blood who had never borne children were examined for their anti-A agglutinin values. Their titers varied from 1:20 to nearly 1:100, with an average of about 1:58. (Many investigators give considerably lower figures for such women.) In a similar series of mothers

10. Polayes, S. H.: The Pathology of Erythroblastosis Fetalis, Proc. New York Path. Soc., January 1942, to be published.

with group O blood who had been delivered of normal children the anti-A agglutinin titer ranged from 1:120 to 1:300, with an average of about 1:215. In contrast to these observations, the bloods of the mothers with blood group O who had been delivered of erythroblastotic infants showed anti-A agglutinin titers ranging from 1:700 to 1:750, with an average of about 1:710 (fig. *A*). Such a marked rise in titer of the anti-A agglutinin must be considered significant in the light of present knowledge of the mechanism of isoimmunization and its role in the pathogenesis of hemolytic anemia of the newborn. In isoimmunization with the Rh factor the Rh antigen of the fetus (inherited from its father), by passing through the placenta, enters the mother's circulation, where it induces development of anti-Rh agglutinins. (The mother's blood, being negative, reacts to the Rh antigen in the same way in which it would react to any other antigen introduced into the body, namely, by building up antibodies against it—in this instance, anti-Rh agglutinins). These agglutinins then retrace the course of the Rh antigen from the mother through the placenta to the fetus and there agglutinate the Rh-positive red cells; hemolysis and hence hemolytic anemia result (fig. B: Rh immunization).

It seems plausible to postulate, therefore, that in a mother with group O blood having a fetus with group A blood exactly the same mechanism may be brought into play. As is shown in the diagram (fig. B), the mother with Rh-negative blood is replaced by the mother with group O blood and the offspring with Rh-positive blood by the offspring with group A blood. The A antigen of the baby (inherited from its father) would enter the mother's circulation via the placenta, and since the mother's blood is of group O, the A antigen of the fetus would induce in the mother an increase in the titer of anti-A agglutinins. (It will be recalled that blood of group O normally has some anti-A and anti-B agglutinins.) By the same token, it may be presumed that a mother with group O blood can be immunized by an offspring with group B or with group AB blood, and also that a mother with group A blood can be immunized by a child with group B blood and a mother with group B blood by a child with group A blo Reports on cases of the lattter types of isoim nization are still wanting, although such stances probably have occurred. As to the mo with group O blood whose child's blood is group A, however (the combination in all of 6 cases), the evidence is nearly complete isoimmunization with the A antigen does oc All 6 patients had characteristic icterus gra neonatorum and fulfilled all the clinical criteria the diagnosis of erythroblastosis fetalis; yet mothers of these babies with group A blood had group O, Rh-positive blood and had anti agglutinin titers of 1:700 or over. It is int esting to note at this point that recently Levine has supplied confirmatory statistical evide that "in this group of mothers there is a hig incidence of matings with men of incompati blood than in the series of mothers whose blo is Rh negative." [5]

CONCLUSIONS

Both of the cases here reported fulfil all t clinical criteria for the diagnosis of eryth blastosis fetalis (icterus gravis neonatorum One patient recovered after a transfusion blood; the other died despite similar thera Postmortem examination of the latter pati showed the anatomic changes in usual eryth blastosis fetalis, including kernicterus, which known to occur almost exclusively in this disea

In both cases the mother's blood group w group O, Rh positive and the child's group Rh positive. The Rh factor and all its kno variants, as well as the Hr antigen, were e cluded as possible immunizing antigens. both cases the anti-A agglutinin in the mothe serum showed a high titer (1:700 and 1:75 a fact which strongly suggests isoimmunizati of the mother with the A antigen of the bab

Although more statistical data are required establish the frequency of such cases, neverthel it is believed that (*a*) isoimmunization by t A and B agglutinogens may occur and th (*b*) erythroblastosis fetalis may result fr A or B isoimmunization by a mechanism si ilar to that already established for the Rh facto

Mr. James McNally Jr. assisted in the technical wo connected with this study.

Prospect Heights Hospital.

HEMORRHAGIC UREMIA

M. G. PETERMAN, M.D.
MILWAUKEE

The appearance in my private service of 2 ses of hemorrhagic uremia at the same time ompted this report. Most of the textbooks on diatrics do not mention hemorrhagic uremia. he few cases cited in the literature involved ults.[1]

REPORT OF CASES

Case 1.—A. N., a 6½ year old girl, was admitted to e Milwaukee Children's Hospital on April 8, 1944. e complaint at entrance was vomiting, which had gun seven days previously and occurred after every al. Four days after the onset diarrhea and abdomal pain after eating developed.

The birth and developmental history of the child were rmal. She had had scarlet fever at 4½ years of age. he family physician reported no complications.

Examination on admission revealed marked anemia, ight edema of the face and numerous petechiae on the ft side of the face. The tongue was dry and coated. here were numerous petechiae on the gums and the ccal surfaces of the cheeks. The blood pressure was 0 systolic and 90 diastolic. The capillary resistance st gave positive results. The urine contained albuin, erythrocytes, a few leukocytes and granular casts able 1). The blood contained 8 Gm. of hemoglobin r hundred cubic centimeters; there were 2,920,000 erythrocytes, 5,900 leukocytes and 64,000 platelets per cubic millimeter of blood; the blood showed a coagulation time of four minutes, a bleeding time of one minute, a Lee coagulation time of five minutes and a clot retraction time of twenty-five minutes. The sedimentation rate (Westergren) was 24 mm. in one-half hour, 45 mm. in one hour and 95 mm. in two hours (table 2). The nonprotein nitrogen content of the blood was 130 mg. per hundred cubic centimeters, the creatinine content 5.6 mg. and the ascorbic acid content 1.5 mg. (table 3).

A diagnosis was made of acute exacerbation of chronic glomerular nephritis (Bright's disease) with thrombopenic purpura. A direct transfusion of 300 cc. of whole blood was given.

April 10: The child had frequent small nosebleeds. There was some edema of the ankles. The platelet count was 44,000, the coagulation time two and one-half minutes and the bleeding time one minute. In the McClure-Aldrich test, with 0.1 cc. of isotonic solution of sodium chloride, the wheal disappeared in thirty-five minutes.

April 11: The wheal disappeared in thirteen minutes; the blood pressure remained at 130 systolic and 82 diastolic. The clot retraction time had increased to one hour and the Lee coagulation time to twelve minutes. The sedimentation rate was 25 mm. in one-half hour, 56 mm. in one hour and 107 mm. in two hours. Liver extract, 1 cc. (15 U. S. P. units) daily, was administered until April 24.

April 12: The child became much more edematous; she had a uremic breath, and new petechiae appeared over the body. A direct transfusion of 300 cc. of whole blood was given. Magnesium sulfate (30 cc. of a 50 per

Table 1.—*Laboratory Data on the Urine of the Patient in Case 1*

Date	Specific Gravity	Reaction	Albumin	Sugar	Acetone	Diacetic Acid	Red Blood Cells	White Blood Cells	Casts
April 8	1.008	Acid	++++	0	0	0	10	3	Coarse, gran., amorph.
10	1.010	Acid	++++	0	0	0	Some	Some	Filled with gran., few hyaline
12	1.013	Acid	++++	0	0	0	5	15	Few gran., amorph.
13	1.010	Acid	++++	0	0	0	10	30	Some gran., occ. hyaline, amorph.
14	1.008	Acid	++++	0	0	0	6	15	Few gran., amorph.
15	.....	Acid	++++	0	0	0	Few	½ pkd	Few gran., occ. hyal.
17	1.006	Neut.	+++	0	0	0	15	Occ.	Occ. gran.
18	1.010	Acid	+++	0	0	0	Many	½ pkd	0
19	1.010	Neut.	++	0	0	0	Many	½ pkd	0
20	1.010	Alk.	+	0	0	0	Many	½ pkd	0
21	1.006	Alk., pink	h t	0	0	0	pk	Many and clumps	Occ. large, waxy
22	1.008	Alk., pink	h t	0	0	0	½ pk	Many and clumps	0
24	1.004	Alk.	+	0	0	0	pk	Many and clumps	0
25	1.010	Neut.	+	0	0	0	¾ pk	¾ pk	0
26	1.008	Alk.	++	0	0	0	40	Pkd and clumps	0
28	1.009	Neut.	++	0	0	0	10	Pkd and clumps	0
29	1.005	Neut.	+	0	0	0	?	Pkd	0
May 1	1.010	Neut., br. red	++++	0	0	0	Pkd fresh	Pkd	Occ. gran., hyal.
2	qns	Neut., br. red	++++	0	0	0	Pkd fresh	Pkd	0
3	1.012	Alk., brown	++++	0	0	0	Many	Pkd	0

1. (*a*) Loeper, M., and Lebret, G.: The Vascular nd Hemorrhagic Form of Uremia, Presse méd. **48**:809 Oct. 15) 1940. (*b*) Gualdi, A.: Hemorrhagic Synromes and Anemia in Chronic Uremia, Policlinico ez. med.) **42**:136 (March) 1935. (*c*) Audoli, L.: he Hemorrhagic Rectocolitis of Uremia, Thesis, Paris, 920.

cent solution) was administered intramuscularly each day until April 16.

April 13. The fundi of the eyes appeared normal. The wheal disappeared in twenty-two minutes. The stools were black and were found to contain blood; they remained so throughout the illness.

April 14: The hemoglobin remained at 8 Gm. and the red blood count at 3,000,000. The platelet count was 100,000. The blood creatinine content was 9.1 mg. per hundred cubic centimeters. Five hundred cubic centimeters of lactose-Ringer (Hartmann's) solution was given intravenously.

April 15: The nonprotein nitrogen content of the blood was 145 mg.; the serum protein was 6.9 mg. One thousand cubic centimeters of lactate-Ringer solution was given intravenously. One cubic centimeter of a preparation of vitamin B complex [2] was given intramuscularly each day until April 21.

April 19: The blood pressure was 116 systolic, 88 diastolic. There was little edema left; the child pale and had a urinous breath. Green iron and ammonium citrates, 0.13 Gm., was given intramuscularly day until April 23. One thousand cubic centimeters 5 per cent solution of dextrose was given intraven

April 30: The edema was almost gone; there many new petechiae on the face. The blood pressure was 120 systolic and 88 diastolic.

April 21: The blood pressure was down to 10 tolic and 76 diastolic. There were many new petechiae on the face. The general condition was poor. The protein nitrogen content of the blood was 175 mg. hundred cubic centimeters and the creatinine co 7.3 mg. The stool gave positive reactions for blood

April 22: The platelet count was 50,000. A transfusion of 360 cc. of whole blood was given.

April 23: The child was more alert; the breath not urinous.

TABLE 2.—*Histologic Observations*

	Leukocytes	Segmented Neutrocytes	Rod and St.	Metamyelocytes	Myelocytes	Eosinophils	Monocytes	Small Lymphocytes	Large Lymphocytes	Myeloblasts	Plasma	Red Blood Cells
April 8.....	5,900	45	12	..	..	1	7	33	1	..	1	2,920,000
	Blood transfusion											
10.....	6,200	27	41	2	1	2	..	23	2	1	1	3,480,000
11.....	.....	..	..	..	..	..	..	..	..	..	..	.....
12.....	Blood transfusion											
14.....	9,400	..	..	..	..	..	..	..	..	..	..	3,000,000
17.....	12,200	..	..	..	..	..	..	..	..	..	..	2,750,000
18.....	Blood transfusion											
21.....	.....	..	..	..	..	..	..	..	..	..	..	2,250,000
22.....	.....	..	..	..	..	..	..	..	..	..	..	.....
	Blood transfusion											
25.....	.....	..	..	..	..	..	..	..	..	..	..	2,400,000
27.....	.....	..	..	..	..	..	..	..	..	..	..	2,170,000
	Blood transfusion											
29.....	.....	..	..	..	..	..	..	..	..	..	..	.....
	Blood transfusion											
May 1.....	8,200	..	..	..	..	..	..	..	..	..	..	2,600,000
	Blood transfusion											
2.....	.....	..	..	..	..	..	..	..	..	..	..	3,100,000
	Roentgen treatment of spleen											
3.....	.....	..	..	..	..	..	..	..	..	..	..	3,250,000
	Blood transfusion and roentgen treatment											

April 16: The edema of the face was more marked, but there was no edema of the extremities. The child had a liquid stool. Five hundred cubic centimeters of 5 per cent solution of dextrose was given intravenously.

April 17: The hemoglobin content dropped to 6 Gm. per hundred cubic centimeters; the number of platelets was up to 192,000. A solution of disodium hydrogen citrate, 4 cc. (containing 0.97 Gm. of the salt), was given every four hours until April 21. Eight hundred cubic centimeters of 5 per cent solution of dextrose was given intravenously.

April 18: A direct transfusion of 300 cc. of whole blood was given. Administration of a liver concentrate with ferrous iron and vitamin B complex,[3] 8 cc. three times a day, was started and was continued to April 24.

April 24: Administration of a preparation of homogenized vitamins,[4] 1 teaspoon three times daily, was started.

April 26: The blood pressure was 140 systolic, 94 diastolic; the nonprotein nitrogen content was mg. per hundred cubic centimeters and the creatinine content 6.5 mg. Blood was oozing from the child's mouth.

April 27: There was oozing of blood from all mucous membranes. The red blood cell count was 2,170,000, the hemoglobin content 6 Gm. per hundred cubic centimeters of blood and the platelet count 28,0 The general condition was poor. An indirect transfusion of 500 cc. of citrated blood was given.

April 28: There was a beginning abdominal ascites. The wheal test required sixty-nine minutes.

April 29: Retrobulbar hemorrhage produced proptosis of the right eye; the left lid was edematous. The liver was enlarged and tender. An indirect transfusion of 500 cc. of citrated blood was given.

May 1: The blood pressure was 140 systolic and diastolic. There was oozing of blood from the vagina.

2. The preparation used was betalin complex (Eli Lilly and Company), 1 cc. of which contains 5 mg. of thiamine hydrochloride, 2 mg. of riboflavin, 75 mg. of nicotinamide, 2.5 mg. of pantothenic acid (as calcium pantothenate) and 5 mg. of pyridoxine hydrochloride.

3. The preparation administered was elixir lexoprin compound, ½ fluidounce (15 cc.) of which represented 1.2 Gm. of ferrous sulfate anhydrous, 1.8 mg. of thiamine hydrochloride and 0.75 mg. of riboflavin with other factors of the vitamin B complex contained in the liver extract.

4. The preparation given was homicebrin, 5 cc. which contains 3,000 U. S. P. units of vitamin A, 0.8 mg. of thiamine hydrochloride, 1.2 mg. of riboflavin, 40 mg. of ascorbic acid and 800 U. S. P. units of synthetic vitamin D.

ed blood cell count was 2,600,000, the hemoglobin . per hundred cubic centimeters of blood, the locyte count 4.5 per cent and the platelet count ; the stool gave a positive reaction for blood. direct transfusion of 500 cc. of citrated blood was

y 2: The blood pressure was 152 systolic and stolic. The urine was now grossly hemorrhagic. was still oozing of blood from the vagina. The lood cell count was 3,100,000, the hemoglobin con- 5 Gm., the platelet count 72,000 and the bleeding ne minute. A roentgen treatment was given to the (setting B, 80 kilovolts, 3 milliamperes, 2 mm. inum filter; 12 inch distance for three minutes at er minute, 45 r, or 10 per cent skin test dose).

y 3: The breath had a urinous odor. The blood ined 214 mg. of nonprotein nitrogen and 9.7 mg. eatinine per hundred cubic centimeters. The plate- unt was 48,000. The sedimentation rate was 35

tained 1,000 cc. and the left 700 cc. of straw-colored fluid slightly tinged with blood.

The heart weighed 160 Gm. The greatest diameters of the valves were as follows: tricuspid, 8.5 mm., pulmonary 4.7 mm., mitral 7 mm. and aortic 3.7 mm.; the left ventricle was 1.5 mm. and the right ventricle 0.3 mm. in thickness. The blood appeared hydremic, and it seemed to be under considerable tension in the venous system. A great deal of blood escaped from the veins under pressure; the total quantity escaping was greater than normal.

The gastrointestinal tract revealed mucosal hemorrhages. There was a great deal of dark red blood in the colon; this extended throughout the colon. This condition was present without any important mucosal lesions and was apparently due to slow oozing of blood.

The pancreas weighed 50 Gm. It was firm, translucent and edematous.

The liver weighed 1,055 Gm. It was 5 cm. below the costal margin. The edges were rounded. It was somewhat translucent from the fluid it contained.

Blood of the Patient in Case 1

mo-bin, m.	Clot Ret.	Lee Coagulation Time	Aniso-cytes	Poikilo-cytes	Actino-cytes	Baso-philic St.	Platelets	Coagulation Time	Bleeding Time	Sedimentation Rate (Westergren)	Prothrombin Time
0	25′	5′	+	+	..	Many	64,000	4′	1	24-45-95	.....
0	...	...	+	+	+	Many	44,000	2½′	1	.....	.....
.	1 hr.	12′	..	..	..	.....	.....	...	..	25-56-107	.....
0	...	...	..	..	..	.....	100,000	...	..	.....	.....
0	...	...	..	..	..	.....	192,000	...	..	.....	.....
0	...	...	..	..	..	.....	.....	...	..	.....	.....
..	...	...	..	..	..	.....	50,000	...	..	.....	.....
5	...	...	..	..	..	.....	.....	...	..	.....	.....
0	...	...	..	..	..	.....	28,000	...	..	.....	.....
..	1 hr.	..	Retic.	..	..	.....	70,000	...	..	.....	Normal
0	...	...	4.5%	..	..	.....	48,000	...	..	.....	.....
5	...	...	..	..	..	.....	72,000	...	1′	.....	.....
0	70′	...	..	..	..	.....	48,000	...	..	35-78-126	.....

in one-half hour, 78 mm. in one hour and 126 mm. wo hours. An indirect transfusion of 500 cc. of ted blood was given. A roentgen treatment was n to the spleen.

ay 4: The child had generalized major convulsions. re were marked nystagmus and Cheyne-Stokes respi-

TABLE 3.—*Chemistry of the Blood in Case 1*

	April 8	11	14	15	21	26	May 3
protein nitro-							
n, mg.....	130	150	158	145	175	145	214
atinine.....	5.6	6.8	9.1	...	7.3	6.5	9.7
orbic acid.....	1.5	...	...	...	...	...	...
al protein.....	...	...	...	6.9	...	...	...

e, Mantoux and Schick tests negative
ol examination—strongly positive orthotoluidine
(test done because of repeated transfusions)

on. The blood pressure was 120 systolic and 88 stolic.

ay 5: An indirect transfusion of 500 cc. of citrated od was given. The patient died.

Autopsy.—The results of a postmortem examination ducted by Dr. G. H. Hansmann were reported as ows:

Gross Observations: The peritoneal cavity contained 0 cc. of clear fluid. The right pleural cavity con-

The right kidney weighed 125 Gm. and the left 145 Gm. Both were mottled red and gray throughout. The cortex was 1 cm. in thickness. The capsules were thin. There was hemorrhage back of each pelvis. There was pus in the left pelvis. Both pelves were reddened from hemorrhage. Scattered through both kidneys were cortical abscesses. The largest abscess measured 1 cm. in diameter. It is estimated that it would take several weeks for such abscesses to form.

The bladder was waterlogged. The mucosa was edematous, and there were hemorrhages into it.

The parathyroids appeared rather large and hemorrhagic.

The bone marrow was gray and hyperplastic.

Bacteriologic Studies: A smear from the kidney revealed many pus cells and clumps of gram-positive cocci. A culture revealed the presence of hemolytic Staphylococcus aureus. A culture of the blood revealed no growth.

Microscopic Observations: The lungs showed some congestion and collapse. There was one small area of capillary bronchitis, and a few air sacs were filled with a bit of exudate.

Examination of the kidneys revealed inflammation of the pelves. There were also somewhat chronic abscesses of the cortex; this interpretation was made because of the presence of a thick layer of granulation tissue about each abscess. In addition, there was diffuse renal disease, as indicated by hyaline thrombi, infarction of the glomeruli, adherence of the glomeruli to Bowman's

capsule, intercapillary fibrosis and inflammatory reaction about the glomeruli. There was also marked tubular atrophy and dilatation. There was some hemorrhage into the tubules. The glomeruli contained some inflammatory cells, and the tubules contained hyaline casts, some of which had inflammatory cells applied on them.

The bladder showed chronic cystitis.

The parathyroid gland showed marked hyperplasia. The sinusoids were distended noticeably with blood.

Diagnosis: The following conditions were included in the diagnosis: staphylococcic septicemia; infectious embolic nephritis; chronic glomerular nephritis; symptomatic thrombopenic purpura; hyperplasia of the parathyroid glands.

CASE 2.—C. S., a 13 year old boy, was admitted to the hospital May 4, 1944. The chief complaint was anorexia.

Nine years previously this boy had had a rash which was diagnosed by the county nurse as "German measles." Several weeks later he became irritable, and abdominal distention developed. The distention lasted one year. The family physician found albuminuria and edema. Since then there had been a recurrence of these symptoms with each infection of the upper respiratory tract. Six weeks before the edema and the distention had

TABLE 4.—*Laboratory Data on the Urine of the Patient in Case 2*

	May 5	May 9	May 10
Specific gravity	1.010	1.010	1.010
Reaction	Acid	Acid	Acid
Albumin	++++	++++	++++
Sugar	0	0	0
Acetone	0	0	0
Diacetic acid	0	0	0
Red blood cells	Occasional	0	0
White blood cells	Occasional	7	Very occasional
Casts	0	0	Very occasionally granular
	No doubly refractive lipoid bodies seen		

May 8, total albumin (24 hr. specimen, total volume 500 cc.) 700 mg. per 100 cc. = 3,500 mg. per 24 hr. specimen

become more marked. The boy had had measles, German measles, mumps, whooping cough, chickenpox and probably scarlet fever.

Examination revealed a pale, sick child with a large, dry, coated tongue, which had many small bleeding points on the under surface. The liver was palpated 7 cm. below the costal margin; the edge of the spleen was palpated. There were signs of fluid in both abdominal flanks. There were large, bleeding external hemorrhoids. The blood pressure was 204 systolic and 160 diastolic. The specific gravity of the urine was 1.010 and remained fixed at that point (table 4). The urine gave a 4 plus reaction for albumin, indicating the excretion of 700 mg. of albumin per hundred cubic centimeters, or 3,500 mg. per twenty-four hours. A very occasional granular cast was seen. No doubly refractive lipoid bodies were found. The blood contained 5.5 Gm. of hemoglobin per hundred cubic centimeters. The red blood cell count was 2,600,000; the sedimentation rate was 15 mm. in one-half hour, 40 mm. in one hour and 78 mm. in two hours (table 4). The nonprotein nitrogen content of the blood was 176 mg. per hundred cubic centimeters, the creatinine 14.2 mg. and the total serum protein 5.9 Gm. (table 5). A diagnosis was made of chronic glomerular nephritis (Bright's disease), hemorrhagic uremia and hemorrhoids. A direct transfusion of 500 cc. of whole blood was given.

May 5: The blood pressure was 164 systolic, 126 diastolic. An indirect transfusion of 300 cc. citrated blood was given.

May 6: There was slight edema of the face; mouth was covered with a white, furry membr sprinkled with blood; the gums were spongy. The domen was distended with fluid. An indirect transfu of 300 cc. of citrated blood was given.

May 8: The red blood cell count was 2,530,000; hemoglobin was 7.5 Gm. per hundred cubic centime The nonprotein nitrogen content was 184 mg. per h dred cubic centimeters, the serum cholesterol 264 and the total serum protein 3.5 Gm., of which 1.71 was albumin and 1.79 Gm. globulin.

May 9: There was bleeding from the mouth and rectum. There was edema of the scrotum. The f showed tortuosity of the arteries; the retinas w normal. The blood pressure was 160 systolic and diastolic. Two hundred cubic centimeters of dextr in lactate-Ringer solution was given intravenously.

TABLE 5.—*Histologic Observations on the Blood of Patient in Case 2*

	May 4	May 8
Leukocytes	9,800	7,600
Seg. neutrophils	88	88
Rods and S. neutrophils	..	1
Eosinophils	..	2
Monocytes	..	3
Small lymphocytes	11	6
Large lymphocytes	1	..
Red blood cells	2,000,000	2,530,000
Hemoglobin, Gm.	5.5	7.5
Anisocytosis	Slight	
Poikilocytosis	Slight	
Achromia	Slight	
Platelets	Plentiful	
Sedimentation rate	15-40-78	

TABLE 6.—*Chemistry of the Blood in Case 2*

	May 4	May 8	May
Nonprotein nitrogen	176	184	.....
Creatinine	14.2	....	.....
Cholesterol	....	264	.....
Alkali reserve	....	....	43.5%
Total protein	5.9	3.5	.....
Kline reaction	0	....	.....
Mantoux reaction (1:1,000 dilution)	0	....	.....
Blood serum:			
Albumin	....	1.71	.....
Globulin	....	1.79	.....
A : G	....	1:1.04	.....

May 10: The ascites had increased. Two hundr cubic centimeters of dextrose in Hartmann's soluti was given intravenously.

May 11: Two hundred cubic centimeters of dextr in lactate-Ringer solution was given intravenously.

May 12: The bleeding continued, and breathing w labored. Four hundred and fifty cubic centimeters dextrose in lactate-Ringer solution was given intr venously.

May 13: The blood pressure was 170 systolic a 128 diastolic, and the child was difficult to arouse.

May 15: The child died in coma.

Autopsy.—A postmortem examination by Dr. G. Hansmann was reported on as follows:

Gross Observations: The right lung weighed 200 G and the left 60 Gm. The right lung was complet

ered with fibrous adhesions. There was frothy coid material in the large bronchi. There were hemhagic areas from 1 to 3 mm. in diameter. On section re was apparent consolidation in these hemorrhagic as. The walls of the bronchioles were thickened. e lung had a reddish brown appearance. The left g revealed a few hemorrhagic areas. There was coid material in the larger bronchi. There was some solidation in the right lung; this condition prevailed roughout the lower lobe.

The large bowel was filled with black, tarry feces. e small bowel was somewhat injected. Some hemorgic areas were noted. There was some dark material o in the stomach.

The right kidney weighed 45 Gm. and the left 75 Gm. e cortex of the left kidney measured 3 mm. and that the right kidney 3.5 mm. The capsule was markedly herent. The kidneys were distinctly shrunken. There s a cystic area 1.5 mm. in diameter on the surface the left kidney. The pelves were greatly dilated. e surface of the kidney was noticeably granular. The ramids were indistinguishable from the cortex. There s an increase in fat about the pelvis.

The aorta revealed moderate atheroma.

Bacteriologic Studies: A culture of the blood revealed aphylococci and Bacillus coli. A culture from the kidy revealed B. coli and Staph. aureus.

Microscopic Observations: The lung contained large lonies of cocci. About the areas in which the cocci re found there was liquefaction of lung tissue with morrhage. About this there was a narrow rim of cent exudate. The lung contained heart failure cells d a great deal of edema.

The kidney revealed extensive pathologic changes; fact, this was the most important finding in the case. e tubules were markedly atrophied secondarily to tensive glomerular injury. Some of the tubules formed rge cysts. Many contained inspissated material. There re no normal glomeruli. Many apparently had dispeared entirely. Those that remained had marked tercapillary fibrosis and attachment to Bowman's psule. A few contained numerous foam cells. The terioles leading to these glomeruli were thickened. here was a considerable amount of interstitial increase fibrous tissue, occasioned by condensation from rophy and proliferative change.

The aorta revealed atheromatous change, as evidenced absence of foci of foam cells and by thickening of e intima.

Diagnosis: The diagnosis was chronic glomerular phritis, early arteriosclerosis, bronchopneumonia and aphylococcic septicemia.

COMMENT

Loeper and Lebret stated that a form of uremia n be identified which is characterized by hemrrhagic processes in all organs and by lesions an analogous nature, although the disturbances ay differ. The vascular and hemorrhagic rocess is always the same, and the toxic cause certain, even though not yet established. They eported the case of a woman of 40 years who ad successively hemorrhages in the retinal, astric, cutaneous, hepatic and cerebral areas. Azotemia was considered significant; toxic amines with an elective vascular effect, particularly tyramine, play a role, and vitamin deficiencies impair vascular trophicity. The blood disturbances—thrombopenia, increased bleeding time and vascular fragility—were considered part of the picture, but they were not considered to be due to vascular alterations. Retention (hyperazotemia and an accumulation of endocrine substances), deficiency of ascorbic acid and intoxication were listed as the main causes of hemorrhage. The treatment suggested was rectal enemas with 0.4 per cent sodium bicarbonate and sodium thiosulfate to increase the alkali reserve.[1a]

Gualdi [1b] reported 4 cases of hemorrhage and anemia occurring in chronic uremia. The patients presented anemia and hemorrhages in the skin, the gingival mucosa and the nose. Only 1 had thrombopenia, and the author suggested that the hemorrhages were due to diffuse capillary vascular alterations.

Audoli [1c] in 1920 described hemorrhagic rectocolitis in uremia and ascribed the bleeding to the complex toxins of uremia and to sudden and frequent transitory hypertension or to continuous hypertension. He stated the well known fact that there is no efficacious treatment for this form of hemorrhage.

Gregory and others [4] recently produced massive gastrointestinal bleeding and azotemia in dogs by giving dog's blood with a stomach tube. They concluded that the syndrome was caused by decreased renal function due to low blood pressure and dehydration or to absorption of digested blood.

My 2 cases of uremia following chronic Bright's disease were characterized by anemia, edema, hemorrhages and hypertension. The patients displayed marked albuminuria, failure of the bone marrow and hyperazotemia. One showed thrombopenia; in the same patient the clotting mechanism as determined by capillary bleeding time, prothrombin time and clot retraction time was normal. Treatment with transfusions of whole blood, liver extract, iron, injections of a vitamin B complex preparation, alkalization and irradiation of the spleen was totally ineffective. Apparently the rapid and relentless progress of the syndrome was not halted in the least. The postmortem observations indicate the fatal character of this syndrome.

411 East Mason Street.

4. Gregory, R.; Ewing, P. L., and Levine, H.: Cause of Azotemia Associated with Massive Gastrointestinal Bleeding, Federation Proc. 2:81, 1943.

CONGENITAL ILEAL ATRESIA WITH GANGRENE, PERFORATION AND PERITONITIS IN A NEWBORN INFANT

STAGED OPERATIONS: OBSTRUCTIVE RESECTION, ILEOCOLOSTOMY AND EXCISION OF EXTERIORIZED ILEUM

ERNEST E. ARNHEIM, M.D.

NEW YORK

Many cases of congenital atresia of the intestine below the duodenum have been reported, but there have been relatively few operative recoveries. The cause of the high mortality is intestinal obstruction, which if not relieved results in necrosis of the bowel and in peritonitis, usually within the first week of life. The outstanding contributions to the subject have been made by Dr. William E. Ladd and his associates,

Since the first operative recovery in a case congenital atresia of the ileum, reported Fockens [2] in 1911, there have been 11 successful operations for jejunal or ileal atresia, 6 of which were recorded by Ladd and Gross. A résumé of case reports is given in the accompanying table. In the experience of Ladd and Gross there were 3 operative recoveries in 6 cases of jejunal atresia and 3 recoveries in 34 cases

Résumé of Reports of Operative Cures of Congenital Atresia of the Jejunum and the Ileum

Case, Year	Reported by	Age, Sex	Clinical Features	Observations at Operation	Operation and Result
1 1911	Fockens [2] (Holland)....	8 days ♂	Vomiting and no bowel movement since birth; abdominal distention and visible intestinal peristalsis	Atresia of midileum; absence of 4 cm. of intestine; no defect of mesentery; intestine much dilated proximal to atresia and collapsed distally	Side to side ileoileostomy; discharged 12 days after operation; well 5 months postoperatively
2 1927	Demmer, F.: Atresia of the Ileum: Ileocecal Resection—Cure, Arch. f. klin. Chir. **147**: 471, 1927 (Austria)	1 hour ♂	Tumor of umbilical cord excised leaving segment of intestinal mucosa on cut surface of umbilical cord; injured intestine necessitated operation	Atresia of lower ileum; absence of 14 cm. of intestine; no defect of mesentery	Excision of base of cecum and ileum distal to atresia; side to side ileocecostomy; discharged 10 days after operation; well 2 years postoperatively
3 1933	Carter, R. F.: Congenital Occlusion of the Duodenum and Small Intestine: A Clinical Consideration with Report of Two Successful Cases, J. Pediat. **2**: 27, 1933	4 days ♂	No bowel movement since birth and fecal vomiting for 12 hours; abdominal distention	Atresia of lower ileum; intestine distended, edematous and congested proximal to atresia and cordlike distally	Side to side ileocolostomy; tube ileostomy; discharged 24 days after operation; well 2 months postoperatively
4 1934	Corkill and Corkill [10]... (Australia)	2 days ♀	No bowel movement since birth and vomiting for 1 day; abdominal distention	Distended intestine ending in midileum, continuing distally as cord 7.5 cm. long; separate closed loop 2.5 cm. long, continuing as cord to cecum; no defect of mesentery	Side to side ileosigmoidostomy; tube ileostomy; discharged 1 month after operation; well 5 months postoperatively
5 1943	Duncan, Wearn, Jackson and Waldron [11] (U. S.)	60 hours (1 mo. premature) ♂	No bowel movement and vomiting since birth; abdomen not distended; roentgenogram of abdomen; gaseous distention of stomach and upper intestine and no gas in remainder of intestine	Dilated jejunum ending blindly 10 cm. below ligament of Treitz: isolated loops of jejunum 4 cm. and 17 cm. long with separate mesenteries; small perforation at distal end of latter plugged by meconium; 2 areas of stenosis at proximal end of distal jejunum	Excision of 17 cm. loop of jejunum and stenotic area of distal jejunum; side to side jejunojejunostomy; prolonged period of biliary obstruction and 2 episodes of severe diarrhea; discharged 109th postoperative day; well 10 months after operation

and their experience is presented in detail by Ladd and Gross in their recent text.[1]

ileal atresia, a recovery rate of 15 per cent in the two groups. Experience has shown that the operative recoveries have been effected by establishment of side to side anastomoses between the parts of the intestinal tract immediately above and below the site of obstruction. Although

Read in part before the Pediatric Section of the New York Academy of Medicine, May 11, 1944.

From the Pediatric Services of Dr. Béla Schick and Dr. Murray H. Bass and the Surgical Service of Dr. Harold Neuhof, The Mount Sinai Hospital.

1. Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1941, pp. 25-43.

2. Fockens, P.: A Case of Congenital Atresia of the Small Intestine Cured by Operation, Zentralbl. Chir. **38**: 532, 1911.

majority of recorded cases the patients have n treated by enterostomy, there is no report his procedure alone having other than a fatal e.

atients with necrosis of the intestine present a more difficult problem, not only because of necessity for excision of the gangrenous part he intestine but because of the desirability of y establishment of intestinal continuity in w of the bad experience with enterostomy. erative management is still further complicated he presence of peritonitis, because anastomotic cedures are contraindicated in this condition. der such circumstances the principles of obctive resection, jejunojejunostomy or ileoostomy, depending on the site of atresia, and ision at the exteriorized intestine seem to me rthy of consideration. They were applied for first time in the following case.

REPORT OF A CASE

istory.—M. J., a girl aged 26 hours, was admitted he Mount Sinai Hospital on April 9, 1942. The nt was born at term by a low forceps delivery. eral hours after birth, the child began to vomit -stained fluid, and abdominal distention was noted the nurse. The vomiting and abdominal distention tinued, and attempts at feeding resulted in more niting. There were two small meconium stools a hours after birth, but enemas later were ineffectual.

hysical Examination.—The infant's temperature was F. She was well developed and well nourished, ghing 7 pounds 12 ounces (3,515 Gm.). The head symmetric in outline; the anterior fontanel was and flat, and the posterior fontanel was open. o marks of the forceps were present over the malar inences of the face. There were no abnormalities the eyes, ears, nose or throat. The heart, lungs, remities and genitalia were normal.

he abdomen was greatly distended; peristaltic waves e not visible. The percussion note was tympanitic, h some dulness in the flanks. No masses were palle, and the liver and the spleen were not felt. Some ness was noted on rectal examination.

Urinalysis revealed no abnormalities. The hemoglo- content was 95 per cent; the leukocyte count was 500, with segmented polymorphonuclear leukocytes per cent, nonsegmented polymorphonuclear leukoes 17 per cent, lymphocytes 22 per cent, myelocytes er cent, eosinophils 1 per cent and basophils 1 per t. The Wassermann test gave negative results.

A roentgenogram of the abdomen, without the use a contrast medium (fig. 1), revealed several loops distended small intestine with fluid levels; no gas s seen in the large bowel. An ovoid mass with a cific periphery, about 5 cm. in its long diameter, s noted in the right lower quadrant.

A Levin tube was passed, and bile-stained fluid was irated. An intravenous infusion of 5 per cent xtrose in isotonic solution of sodium chloride was inistered for two hours when operation was perrmed.

First Operation (April 9, 1942).—With the patient der ether anesthesia, a 3 inch (7.5 cm.) right rectus scle-splitting incision was made. A large amount light brown, odorless fluid was removed by suction from the peritoneal cavity. The operative site was filled with a greatly distended loop of small intestine, dark blue and covered by a thin film of vascular fibrinous adhesions. Retraction of the mesial side of the wound revealed similar-appearing intestinal loops filling the lower part of the abdomen and a large collection of dark brown fluid.

In order to visualize the site of intestinal obstruction, the loops of distended and discolored intestine were allowed to extrude from the peritoneal cavity. As the infant strained, an escape, in large amounts, of yellow, odorless meconium was seen within the peritoneal cavity, without manipulation of the part of the intestine from which the meconium escaped. A rapid exteriorization of the ileum was performed, in the course of which a volvulus of the ileum was untwisted and some inflammatory adhesions in the right lower quadrant were divided. After exteriorization of the ileum, the observations were as follows:

Fig. 1.—Roentgenogram of the abdomen (without the use of a contrast medium) taken on the patient's admission to the hospital, showing several loops of distended small intestine with fluid levels; no air is seen in the large bowel; an ovoid mass with a calcific periphery is seen in the right lower quadrant.

A loop of ileum, about 25 cm. in length and 4 cm. in diameter, was reddened, thickened and lusterless; much of the serosal surface was covered by thick fibrinopurulent exudate. When traced distally, the loop of the intestine was found to end as a blind sac. A band of fibrous tissue about 1.5 cm. in length, projecting from the mesenteric border of the intestine, represented the site of atresia in the terminal part of the ileum. The end of the blind sac of intestine proximal to the atresia, over an area about 3 cm. in diameter, was necrotic and of the consistency of tissue paper. In this area of gangrene there was a perforation about 3 mm. in diameter.

The mesentery of the blind sac of gangrenous and inflamed ileum was divided, and the mesenteric vessels were ligated. One small Payr clamp was applied to the intestine proximal to the site of the ligated mesentery, and a second small Payr clamp was applied to the collapsed intestine about 2 cm. distal to the site of atresia. The section of intestine between the clamps was removed with the knife treated with phenol. The resected intestine represented the area of necrosis and of most severe inflammation; an additional segment of inflamed intestine covered by exudate was not removed. The proximal and distal loops of ileum, closed by clamps, were left outside the peritoneal cavity on the abdominal wall. After application of about 0.5 Gm. of sulfanilamide powder in the operative site, the abdominal wall was closed with through and through sutures of silk, liberal drainage being allowed into the peritoneal cavity around the exteriorized intestine by pieces of rubber dam. Iodoform gauze was placed around and between the loops of intestine. To avert damage to the thin intestinal wall, continuous silk sutures were applied over the Payr clamps, the clamps withdrawn and the sutures tied. A transfusion of 50 cc. of citrated blood, supplemented by 5 per cent dextrose in isotonic solution of sodium chloride, was administered throughout the operation.

Fig. 2.—Photograph of the resected segment of small intestine, showing the site of atresia of the ileum with the proximal and distal loops of intestine, and the area of necrosis with a probe passing through the site of perforation.

Pathologic Report.—The specimen (fig. 2) consisted of a resected segment of small intestine 17 cm. in length. A dilated proximal loop, 13 cm. in length, was separated from a contracted distal loop, 2.5 cm. long, by a fibrous band 1.5 cm. in length. The proximal loop was 4 cm. in diameter, the distal loop 6 mm. and the atretic band 3 mm. The dilated intestine was markedly injected. Proximal to the area of atresia the wall of the intestine was greenish black; a perforation was present in the area of necrosis. The mucous membrane proximal to the necrotic region was red and injected, and the muscularis, especially near the obstruction, appeared somewhat thickened. The mucosa of the distal (contracted) loop was normal in appearance. On microscopic examination the area of atresia was found to consist of fibrous tissue with deposits of calcium; the serosal surface of the atretic ileum showed a few hairs surrounded by a foreign body reaction.

Bacteriologic Report.—A culture of the peritoneal fluid was reported to contain Bacillus coli.

Course.—The infant's condition remained good for sixty hours. Abdominal distention was controlled by an indwelling Levin tube and by gastric siphonage (Wangensteen), 100 cc. of bile-stained fluid b drained off during this time. The wound was cl and the exteriorized ileal loops were slightly discolo The infant's highest temperature during this pe was 100.6 F. An intravenous infusion of 5 per c dextrose in isotonic solution of sodium chloride in distilled water was given continuously and was s plemented by small doses of sulfathiazole sodi Oxygen was administered continuously in an oxy cubicle (fig. 3).

Second Operation (April 12, 1942).—Sixty ho after the first operation, with the patient under et anesthesia, a 3 inch (7.5 cm.) left rectus musc splitting incision was made. There was a small amo of light brown, odorless fluid in the peritoneal ca at the operative site. The loops of small intestine the region appeared normal and slightly dilated. the lower part of the abdomen was approached, small intestine became more dilated, and there w fibrinous adhesions between loops of bowel. The p tion of inflamed segment of ileum which had not b resected at the first operation was isolated. The int tine was dilated and reddened, but less so than at previous operation, when it was covered with fibri purulent exudate. Identification was aided by visu zation of the rubber dam drains coming through original operative wound. The distal loop of exteri ized ileum was not identified. The posterior peritone in the lower part of the abdomen, near the origi operative site, was inflamed.

The mesentery of the proximal loop of ileum w divided about 3 inches (7.6 cm.) from the site of teriorization of the bowel, and the mesenteric ves were ligated with fine silk. Two small Payr clam were applied to the ileum at the margins of the divi mesentery, and the bowel was cut through with cautery. The ends of the divided ileum were clo in an aseptic manner by continuous sutures of fine s passed over the clamps. The transverse colon was t identified. A side to side isoperistaltic anastomosis w made between the proximal loop of ileum and transverse colon (fig. 4). There was great dispar in size between the dilated ileum and the collap colon; the ileum was about 4 cm. in diameter and colon about 7 mm. The anastomosis was effected using interrupted fine silk sutures for the seromuscu layers and continuous 00000 chromic catgut for mucous membrane. Sulfanilamide powder was sc tered over the site of the anastomosis and over ileum proximal to the anastomosis. The abdom was closed with through and through sutures of si At the end of the operation both loops of exterioriz ileum on the abdominal wall were opened by remov of the silk sutures.

Course.—During the first three postoperative da the infant's good condition was maintained by dai transfusions of 50 cc. of citrated blood, intraveno infusions of dextrose solution and isotonic solution sodium chloride, gastric siphonage and oxygen thera Intravenous infusions and gastric siphonage were d continued on the third day. Feedings of breast m were started on the third day, and normal bow movements were noted on the following day. On t ninth day the infant was removed from the oxyg cubicle. At this time thin, bile-stained drainage w noted from a small perforation in a loop of intestine a herent to the mesial side of the first operative wou The exteriorized loops appeared normal.

During the second and third postoperative we there was a loss of weight to 5 pounds 15 oun (2,693 Gm.), despite adequate nourishment and norm bowel movements. This was associated with second

ia and dehydration; the hemoglobin varied from r cent to 74 per cent, and the plasma protein was h as 7.49 Gm. per hundred cubic centimeters. Four fusions of citrated blood of 50 cc. each and fluids nistered parenterally were given during the second hird weeks to combat the anemia and the dehy-on.

the eighteenth postoperative day there was a rise mperature to 103 F. and an increase in respiratory o between 60 and 80 per minute. This was asso-d with cyanosis and with rhonchi in both lungs. episode of bronchiolitis lasted for one week was successfully treated with oxygen and sulfa-ole. A roentgenogram of the abdomen, without sponse to treatment was good, and one week after the episode of diarrhea the weight was 5 pounds 15 ounces (2,693 Gm.) and the hemoglobin content was 89 per cent.

On the fifty-fifth postoperative day the small perforation of the bowel adherent to the wound closed spontaneously. The exteriorized, blind, proximal loop of intestine presented some openings in the anterior wall and a partial prolapse of the posterior wall.

On the seventy-fifth postoperative day the hemoglobin content was 64 per cent, and a transfusion of 75 cc. of citrated blood was given. After the transfusion there was a gradual gain in weight to 7 pounds 2 ounces (3,232 Gm.) on the eighty-first day. An addi-

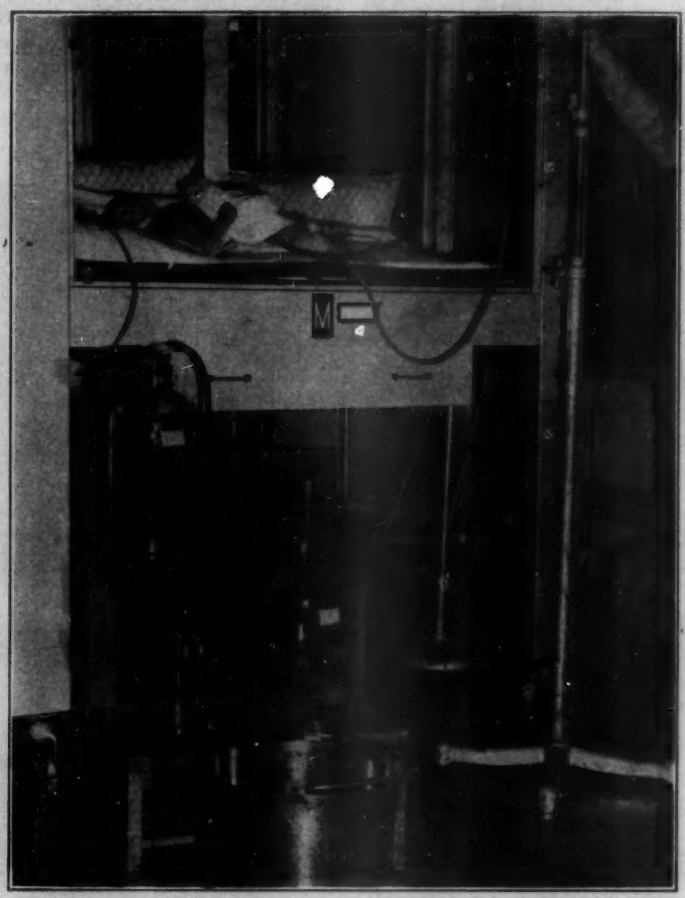


Fig. 3.—Photograph of the infant in the interval between the stages of obstructive resection and ileocolos-y, showing fluids administered parenterally through a small vein exposed at the ankle, gastric siphonage tained by suction through bottles and oxygen let in by a vent in the cubicle. (The infant is exposed for photograph.)

use of a control medium, on the twenty-eighth perative day (fig. 5) revealed gas in several loops wel, most of which represented large intestine not ly distended; a small amount of gas was noted e rectum. The ovoid mass with a calcific periph-noted in the preoperative roentgenogram, was not nt. Some loss of fluid continued from the small ration of the bowel, but the stools were normal, there was a gradual gain in weight to 7 pounds 5 Gm.) during the sixth postoperative week.

n the forty-fourth postoperative day severe diar-began, lasting four days and resulting in a loss eight to 5 pounds 3 ounces (2,353 Gm.). The hea was treated with a transfusion of citrated d and fluids administered parenterally. The re-tional transfusion was given on the eighty-sixth post-operative day, and the hemoglobin content rose to 87 per cent.

From the twelfth to the fifteenth postoperative week there was no appreciable progress. The appearance of the infant was striking (fig. 6). The distended abdomen was in marked contrast to the wasting extremities and chest wall. The weight remained stationary at about 7 pounds (3,175 Gm.), despite an adequate caloric and vitamin intake. The stools numbered from three to six per day and varied in consistency from watery to normal. Some stools were bulky and foul, and a chemical analysis of one of these revealed 56.5 per cent fat content. Various formulas, based on evaporated and protein milk, did not influence the

weight or the stools. Examination of the duodenal secretion for pancreatic ferments revealed the presence of diastase and a trace of trypsin.

During the fifteenth postoperative week feedings of skimmed milk, with an additional 10 per cent honey and banana, were begun; 2 Gm. of pancreatin was added to each feeding. There was an immediate gain in weight, but during the following week fever and diarrhea were constant. Anemia (hemoglobin 50 per cent) was corrected by two transfusions, which raised the hemoglobin to 80 per cent. When the child had been on the skimmed milk regimen for seven weeks (and had been given pancreatin for four weeks) the stools became more normal in consistency and usually numbered from one to three a day, but there were occasional episodes of diarrhea. A chemical analysis of the stool revealed 5 per cent fat content. The weight increased steadily to 9 pounds 3 ounces (4,167 Gm.) in the twenty-third postoperative week.

An examination of the duodenal secretion for pancreatic ferments during the twenty-second postoperative week (nineteen days after administration of pancreatin had been discontinued) revealed a trace of diastase and an absence of trypsin. Applesauce and cereal containing a casein hydrolysate were then added to the skimmed milk (800 cc. per day). In order to promote the metabolism and assimilation of protein nutramigen (125 Gm. in 960 cc. of water, divided into six feedings) was made the basis of the formula, to which were added pea puree (50 Gm.), cerevim (25 Gm.), pancreatin and vitamins. Saccharated ferrous carbonate was given for mild secondary anemia. The infant was discharged on the one hundred and ninetieth postoperative day (aged 6 months), weighing 9 pounds 8 ounces (4,309 Gm.).

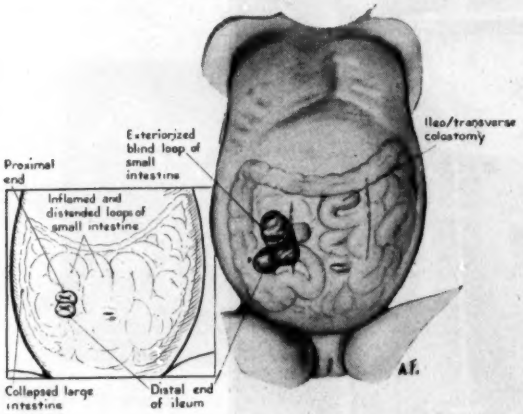

Exteriorized blind loop of small intestine
Ileo/transverse colostomy
Proximal end
Inflamed and distended loops of small intestine
Collapsed large intestine
Distal end of ileum

Fig. 4.—Drawing of the abdomen illustrating the status of the intestines after completion of the obstructive resection and the ileotransverse colostomy. Inset showing the intestinal tract between the operative procedures.

Follow-up examination, when the child was 10 months old, revealed a continued improvement since her discharge. The diet had been low in fat, the formula consisting of buttermilk, water and dextrimaltose; added solid foods included vegetables, meats, cereals, egg, fruit-flavored gelatin, sweet potato and cottage cheese. There were three stools a day, the first of which was formed and the succeeding ones loose but normal in color and nonodorous. The weight was 14 pounds 8 ounces (6,577 Gm.). The abdomen was slightly distended, the loops of exteriorized ileum presenting in the right lower quadrant.

Second Admission.—The child was readmitted to Mount Sinai Hospital on May 25, 1943, at the age 13 months, for management of the exteriorized ile There had been no change in the character of stools. The child's temperature was 99 F. She well developed and well nourished; she was able stand, had eight teeth and weighed 22 pounds (9 Gm.). The abdomen was somewhat protuberant, senting firm scars 6 and 9 cm. in length and the teriorized loops of ileum. The proximal loop some openings on the anterior wall and a partial lapse of the posterior wall.

Physical Examination.—Urinalysis revealed no normalities. The hemoglobin content was 66 per c the leukocytes numbered 8,600. After transfusion 150 cc. of citrated blood the hemoglobin rose to 82 cent. A dextrose tolerance test revealed a fas blood sugar content of 40 mg. per hundred cubic timeters followed by elevation to 115 mg. at one-h hour and 140 mg. at one hour and a fall to 25 mg two hours.

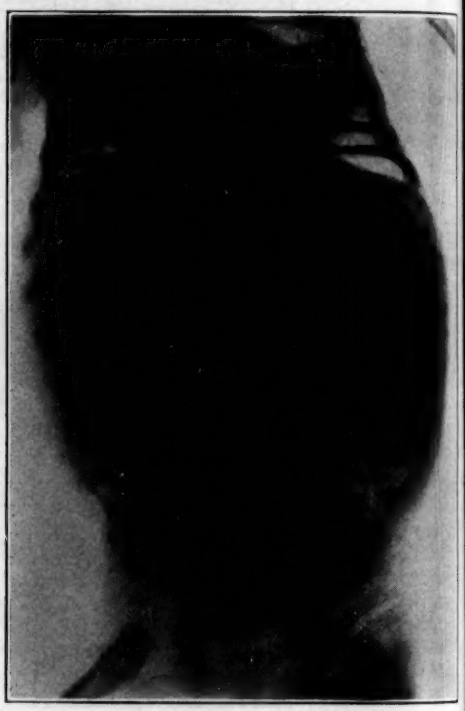


Fig. 5.—Roentgenogram of the abdomen (without use of a contrast medium) taken twenty-eight d after the operation, visualizing loops of large intes filled with air.

Third Operation (June 11, 1943).—With the c under ether anesthesia, cutaneous incisions were m encircling the exteriorized bowel. The free perito cavity was entered. There were relatively few ad sions, and presenting loops of small intestine w smooth and glistening.

The exteriorized loops of ileum were identified wi the peritoneum and were found to be approximated each other by thin adhesions. Attention was f directed to the proximal loop, which extended upw toward the liver for about 13 cm. It was about 0.8 c in diameter and of normal thickness. After the bl end was isolated, the proximal loop of ileum was e cised after the mesentery had been divided betw clamps and the mesenteric vessels ligated with fine s

e distal loop of ileum was traced downward for tance of about 8 cm. and was found to come to nd end about 2 cm. from the cecum; thus a second ia was demonstrated. The distal loop was about . in diameter, but the distal 2 cm. attached to the n was narrow and obliterated. The cecum and ppendix were normal in size, appearance and loca- The distal loop was excised to the cecum after ion and ligation of the mesentery. Operation was nued to eliminate adhesions of an extremely dis- d loop of small intestine to the parietal peritoneum the capsule of the liver.

ter application of about 2 Gm. of sulfanilamide der in the peritoneal cavity, the wound was closed through and through sutures of silkworm gut.

completely obliterated cordlike structure. A small fragment of mesentery, containing a number of small lymph nodes, was attached to the distal segment of ileum.

Course.—A transfusion of 250 cc. of citrated blood was administered during the first postoperative day, after which an intravenous infusion of 5 per cent dextrose in isotonic solution of sodium chloride and in distilled water was administered continuously until the third day. The temperature was 102.4 F. on the second postoperative day and normal thereafter. The bowels moved on the third day. The wound healed by primary union. The child was discharged sixteen days after operation; a formula of protein milk with 5 per cent dextrimaltose and additional potato, vegetables and beef were prescribed.

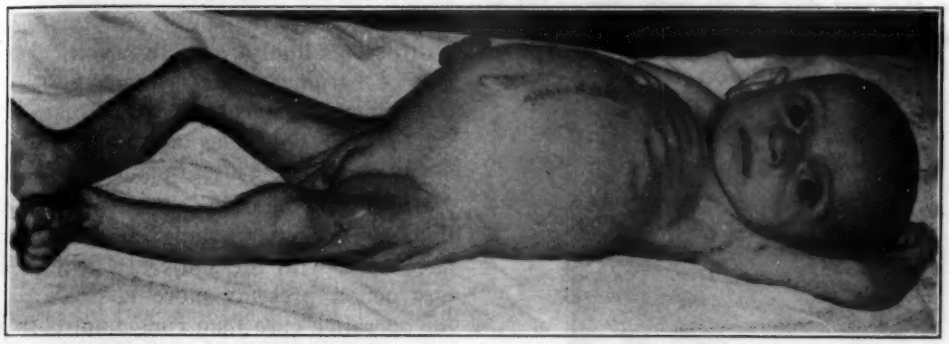


Fig. 6.—Photograph of the infant taken during the twelfth postoperative week, showing the wasting extremities chest wall and the distended abdomen with the exteriorized loops of small intestine.

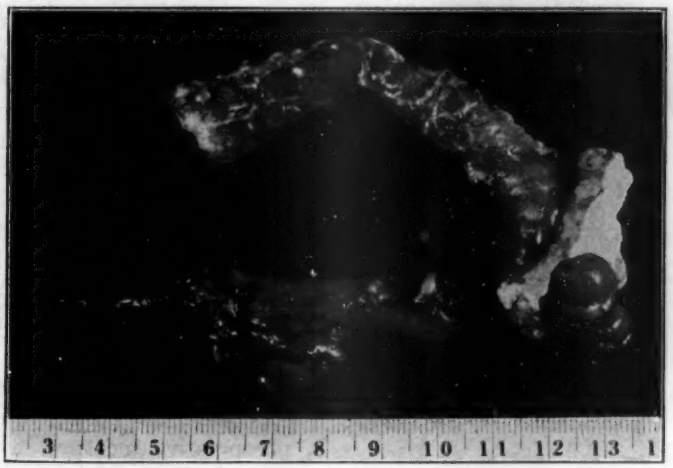


Fig. 7.—Photograph of the excised blind loop of small intestine and the distal loop of ileum terminating in a nd atresia attached to the cecum (not shown). A segment of skin and subcutaneous tissues surrounds the eriorized openings of both loops.

e child's condition at the termination of the opera- was good.

athologic Report.—The specimen (fig. 7) consisted resected portion of ileum 23 cm. in length. Two mas, surrounded by a small piece of skin, were nd 13 cm. from the proximal line of resection. The ximal segment measured 1.5 cm. in diameter ened), was of normal thickness and contained a ll amount of mucoid fluid. The serosal surface wed some fibrous adhesions, and the mucosa was . The distal segment of ileum was 10 cm. in gth. The proximal 8 cm. of this loop was 2 cm. circumference (opened); the distal 2 cm. was a

Follow-up examination when the patient was 20 months old (fig. 8) revealed continued improvement with a normal diet. The weight was 25 pounds (11,340 Gm.) and the height 32 inches (81 cm.). The child's motor behavior was normal; she walked with occasional support. There were three stools a day, the first formed and the succeeding ones loose but of normal color.

At follow-up examination when the child had reached the age of 2 years and 3 months the weight was 28 pounds (12,700 Gm.) and the height 35 inches (89 cm.). There was one normal stool a day. The wounds were firmly healed.

At the time of this report, the patient's age is 2 years and 6 months, the weight is 29 pounds (13,154 Gm.) and the height is 36 inches (91 cm.). There is one normal stool a day, and the abdomen is not distended. Mental and physical development is normal, and the patient is an alert, happy child.

COMMENT

Early recognition of atresia of the intestine is based on a prompt evaluation of the clinical manifestations. Vomiting is the primary symptom, and its inception is usually related to the location of the obstruction, but vomiting is often noted on the first day of life with atresia below the duodenum. The vomitus is bile stained and later becomes intestinal. The stools are usually smaller, and they do not contain any bile pigment. The presence and the degree of abdominal distention depend on the location and the duration of the obstruction; there may be visible and audible peristaltic movements. As the obstruction continues, the usual changes secondary to dehydration are noted: poor turgor of the skin, decreased urinary output, evidences of hemoconcentration and fever. With infarction and perforation of the intestine these changes become more marked, and finally, usually in the first week of life, evidences of shock and/or peritonitis are present.

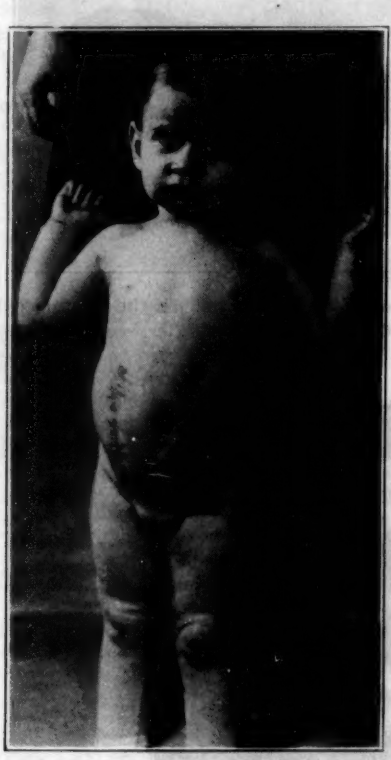


Fig. 8.—Photograph of the child at the age of 20 months, showing good nutrition and the operative scars.

Roentgen examination of the abdomen is decisive aid in diagnosis. In this connectio statement of Ladd and Gross [1] deserves qu tion: "Any child who continues to vomit dur the first day or two of life while being fe normal amount of breast milk or a reasonable f mula should be investigated by roentgenogra for intestinal obstruction." Roentgenograms the abdomen taken without the use of a cont medium are often informative.[3] The la amounts of air swallowed by a newborn ini distend the intestine proximal to the obstruct in marked contrast to the absence of gas in distal portion of the bowel. With more advan obstructions fluid levels are visible. Administ tion of barium sulfate is unnecessary and ti consuming. The barium may be aspirated i the lungs by vomiting or may subsequently struct the intestine after operation.

Congenital duodenal obstruction is not wit the scope of this communication, and many pap have dealt with the subject. Intrinsic duode obstruction presents more favorable conditi for operative cure than congenital obstruction the jejunum or the ileum—a large proportion incomplete obstructions (stenosis), absence multiplicity of lesions or of complicating v vulus and a less virulent bacterial flora—a some 40 operative recoveries have been report Other causes of intestinal obstruction in the fi week of life are congenital malformations of t anus and the rectum, malrotation of the intesti and meconium ileus. The anomalies of the an and the rectum are easily diagnosed. The diffe entiation of atresia, stenosis and malrotation the intestine is aided by Farber's test [4] for t presence of cornified epithelial cells in the m conium. It is most important to establish t more general diagnosis of intestinal obstructi so that early operation may be carried out.

Operation must be supplemented by adequ preoperative and postoperative management. a preoperative measure, which should be co tinued during and after operation, parente administration of fluids is of the greatest impo tance. The intake of fluids should be maintain at 2½ or 3 ounces per pound (150 to 180 cc. p kilogram) of body weight per twenty-four hou This should be increased to compensate for lo

3. Wangensteen, O. H.: The Therapeutic Probl in Bowel Obstructions, Springfield, Ill., Charles Thomas, Publisher, 1937, p. 173. Ladd and Gros Cohen, P.: Congenital Intestinal Obstruction w Special Reference to the Value of the Plain Roentge gram for Diagnosis, Am. J. Dis. Child. **61**:135 (Ja 1941.

4. Farber, S.: Congenital Atresia of the Alimenta Tract: Diagnosis by Microscopic Examination Meconium, J. A. M. A. **100**:1753 (June 3) 1933.

body fluids by vomiting or gastric siphonage. the case reported, fluids were administered tinuously by the intravenous route for four s. For this purpose a vein at the ankle was osed by a small incision and a Karelitz dle [5] tied in place. Infants are easily over- rated, and the amount and speed of adminis- ion of the fluid must be carefully regulated. ferably by a dropper arrangement in the in- venous set. If there are difficulties with the avenous infusions, isotonic solution of sodium oride can be given by the subcutaneous route. l feedings are withheld in the early post- rative period. If hypoproteinemia or shock present, blood plasma should be administered avenously. Transfusions of whole blood from uitable donor are indicated in the presence of mia; thirteen transfusions of whole blood e administered to the infant whose case is orted here.

The feeding problem may be a difficult one, l in its management the supervision of a iatrician is necessary; this is illustrated in postoperative course of the patient described. vere diarrhea has been recorded in cases other n the present one, and in those cases formulas ed on protein milk were beneficial; in this e nutramigen was found useful to assist in the tabolism and assimilation of protein. Intestinal sorption was considerably deranged in this ant by the excision of 17 cm. of ileum and the etracking of an additional 23 cm., in the new- n period. In some ways the type of nutritional lure simulated celiac disease, but pancreatic ylia was not proved, and the subsequent nor- l development of the child is additional evi- nce that the cause of the nutritional failure was emporary interference with intestinal absorp- n. Vitamins should be added to the diet of se infants in amounts considered essential for ants of their age. Infants in the newborn iod may have a low concentration of pro- ombin in the blood, and it is advisable to ad- nister vitamin K in this period.

Other useful measures in the management of se infants are: passage of a Levin tube and intenance of gastric siphonage (Wangensteen) long as intestinal or biliary drainage contin- s; inhalation of oxygen supplied through a tent cubicle, and administration of sulfonamide mpounds for postoperative infections.

As was stated in the introductory remarks, ngenital atresia of the intestine should be eated by establishment of a side to side anas- mosis between the parts of the intestinal tract mediately above and below the site of ob- struction. The uniformly bad results with enterostomy alone as an operative procedure in this condition should discourage any such operation. It would seem that infants in the newborn period do not tolerate the dehydration secondary to loss of fluids and intestinal juices from enterostomy. The longest survival with enterostomy alone was reported by Smith [6] in a case of stenosis of the intestine, in which the infant lived for forty-one days after ileostomy. However, it is feasible to establish an enterostomy as a procedure preliminary to anastomosis or to combine it with an anastomosis, although the latter procedure would appear to be unnecessary. In the 5 case reports summarized in table 1 it is to be noted that ileostomies were performed in combination with anastomoses in 2 cases. In a paper on the subject, in 1933, Ladd [7] referred to a cure in a case of atresia of the ileum associated with a volvulus and with peritonitis, in which an ileostomy was performed after excision of a 12 cm. loop of ileum, followed, twenty-three days later, by an ileotransverse colostomy. In their monograph Ladd and Gross [1] mentioned 2 infants with atresia of the ileum "who were first treated by ileostomy and who lived for three and four weeks respectively, at which time anastomosis of the proximal and distal loops was undertaken. Both of these children died, but they were temporarily relieved of their obstruction and might have been cured if the anastomosis had been attempted at an earlier date." These authors suggested that an ileostomy might be performed on patients with congenital obstructions below the jejunum, followed by an anastomosis in two or three days.

Side to side anastomoses in infants with congenital atresia of the intestine present problems in operative technic because of the disparity in size between the distended proximal loop and the collapsed and small distal loop. Clogg [8] and Webb and Wangensteen [9] have suggested dilation of the distal loop with air. Ladd and Gross insert a small urethral catheter in the distal loop after it is opened; the catheter is removed before completion of the anterior row of sutures. Fine suture material on atraumatic needles is of great assistance in the operative technic.

Since congenital atresias of the intestine are occasionally multiple, it is necessary to explore the intestine so as not to overlook additional

5. No. 542 L.N.R. Becton, Dickinson and Co.

6. Smith, G. K.: Congenital Intestinal Occlusion, M. J. Australia **2**:685, 1932.

7. Ladd, W. E.: Congenital Obstruction of the Small Intestine, J. A. M. A. **101**:1453 (Nov. 4) 1933.

8. Clogg, H. S.: Congenital Intestinal Atresia, Lancet **2**:1770, 1904.

9. Webb, C. H., and Wangensteen, O. H.: Congenital Intestinal Atresia, Am. J. Dis. Child. **41**:262 (Feb.) 1931.

atresias. If multiple atresias are present, the intestine below the lowermost atresia must be selected for the distal loop of the anastomosis. Ladd and Gross [1] advise against excision of the isolated loops of intestine, and this counsel has been followed in the cases of Corkill [10] and of Duncan, Wearn, Jackson and Waldron.[11]

Necrosis of the intestine with peritonitis may follow the formation of a volvulus of the distended blind end around the atretic cord, or perforation may be incidental to necrosis of the bowel secondary to occlusion of the arteries from overdistention of the intestine. It is of interest in this case that B. coli was cultured from the peritoneal fluid at operation, twenty-eight hours after birth, and that the appearance of the peritoneum was that usually associated with advanced secondary peritonitis. Ladd [7] reported 8 instances of perforation in a series of 40 cases of congenital obstruction of the small intestine, and these occurred in obstructions below the duodenum; the recovery in a case of atresia of the ileum with peritonitis has been previously mentioned. The case reported in this communication presents the second such cure. In both cases impairment of circulation necessitated intestinal resection. In Ladd's case [12] this was combined with an ostomy, followed, twenty-three days later, by ileotransverse colostomy without exclusion; subsequent course was complicated by episo of intestinal obstruction and difficulty in clos of the ileal fistula. In order to obviate the pr lems associated with enterostomy which h been previously outlined, the plan followed in case reported here was utilization of the princi of obstructive resection, followed sixty hours l by an ileotransverse colostomy with exclus and finally, thirteen months later, by excision the exteriorized intestine.

10. Corkill, T. F., and Corkill, H. K.: Congenital Atresia of the Ileum, Australian & New Zealand J. Surg. **3**:352, 1934.

11. Duncan, P. A.; Wearn, F. S.; Jackson, H. F., and Waldron, W. S.: Successful Surgical Treatment of Multiple Atresias (Aplasias) of the Small Intestine in a Premature Infant, J. A. M. A. **123**:764 (Nov. 20) 1943.

12. Ladd, W. E.: Personal communication to author.

SUMMARY

A case of congenital atresia of the ileum w gangrene, perforation and peritonitis in a n born infant is reported. This is the twel reported cure of atresia of the intestine below duodenum and the second cure of a patient this condition when perforation and peritoni had supervened. The surgical plan was that staged operations, consisting of obstructive res tion, ileocolostomy and excision of the exteri ized ileum.

Dr. Béla Schick and Dr. Murray H. Bass coopera in the treatment of this patient. Dr. Louis H. Bar berg cared for the child after her discharge from hospital and Dr. Sadao Otani made the photogra preparations of the specimens.

16 East Ninety-Sixth Street.

Correspondence

PYGOPAGUS TWINS

the Editor:—I have just read the excellent paper Dr. Helen Dawson on the anatomic abnormalities set of pygopagus twins, published in the AMERICAN NAL OF DISEASES OF CHILDREN for December 1944. r the sake of completeness, I wish to take exception e statement made by Dr. Dawson, "A number of pagus twins have been described in the literature, nfortunately no descriptions of complete dissections available." I should like to call attention to a paper en by the late Dr. E. R. LeCount and me and ished in the *Archives of Pathology and Laboratory cine* (**3**:171-192 [Feb.] 1927) giving a complete rt on the observations at necropsy on the famous pagus twins, Rosa and Josepha Blazek. Although Dr. Dawson mentioned these twins in her paper and gave several references to them, she apparently had failed to see our paper, which summarizes the literature on these pygopagus twins and describes the anatomic abnormalities.

It might be of interest to note that although the autopsy on the Blazek twins, which was performed by Dr. LeCount, was limited to a pelvic and low abdominal incision, this intrepid pathologist was able to remove practically all of the organs, including the thyroid glands, from both of these sisters. I believe that Dr. Dawson and others reviewing the subject of pygopagus twins will therefore be interested in this reference.

M. A. PERLSTEIN, M.D., Chicago.

185 North Wabash Avenue.

News and Comment

hysicians Certified as Specialists by the Amer- Board of Pediatrics.—The following physicians certified as specialists by the American Board of iatrics after the examinations in New York, Dec. 1944:

pt. Fred Harold Allen, Bradley Field, Conn. (for- y Holyoke, Mass.); Dr. Erna Goettsch Anderson, on; Dr. Alta Ashley, Wellesley Hills, Mass.; Dr. uel Righter Berenberg, New York; Dr. Ubaldo L. ari, New York; Dr. James T. Cameron, Quincy, s.; Dr. David Jerome Cohen, Meriden, Conn.; Dr. ard Michael DiTolla, New York; Dr. Hania licka Ehlers, Baltimore; Dr. Helene Eliasberg, New k; Lt. Marcus E. Farrell, Sampson, N. Y. (for- y Clarksburg, W. Va.); Dr. William H. Fost, eville, N. J.; Dr. Paul Freud, Forest Hills, N. Y.; Benjamin Goldsmith, Flushing, N. Y.; Dr. Jacob nblatt, Stamford, Conn.; Dr. John Robert Heafey, h Norwalk, Conn.; Lt. Bernard John Houston, oklyn (formerly Philadelphia); Dr. Leo Jenkins, nyside, Long Island, N. Y.; Lt. Chris Peter Kat- pes, Washington, D. C. (formerly Rochester, N. Y.); t. Michael C. Kemelhor, Staten Island, N. Y.; Dr. ea D. Kessler, Upper Darby, Pa.; Dr. Jules I. n, Morgantown, W. Va.; Dr. Max Landsberger, alo; Dr. Lucille Liberles, Baltimore; Capt. Bernard Lilien, Montgomery, Ala. (formerly Irvington, N. J.); J. Philip Lombard, Brooklyn; Dr. Anthony J. ia, New York; Capt. W. M. McFadden, Balti- e (formerly Germantown, Pa.); Dr. Mark Mohr, oklyn; Capt. James P. Morrill, San Antonio, Texas; Gertrud Clara Reyersbach, Boston; Dr. Robert A. ess, Philadelphia; Dr. Bernard A. Schmierer, Brooklyn; Dr. Austin Howard Schoen, Brooklyn; Dr. Lawrence M. Shapiro, New York; Dr. Dorothy Louise Shindel, Philadelphia; Dr. Gertrude Slater, Stamford, Conn.; Dr. Margaret H. D. Smith, Baltimore; Dr. Anthony Gerard Stigliano, Brooklyn; Lt. Caleb O. Terrell Jr., Newport (Melville), R. I. (formerly Fort Worth, Texas); Dr. Jean Wells, West Hartford, Conn.; Dr. Eleonore C. Zaudy, Boston and Jamaica Plain, Mass.

GENERAL NEWS

Application Fee for American Board of Pediatrics.—The application fee for the American Board of Pediatrics will be raised to $75 on May 1, 1945.

PERSONAL NEWS

Promotion for Dr. Alfredo Casaubon.—Dr. Alfredo Casaubon has been advanced from adjunct professor to associate professor at the Clinica Pediátrica y Puericultura de la Facultad de Ciencias Médicas de Buenos Aires.

According to the October 1944 issue of the *Archivos Argentinos de Pediatría* the eminent French pediatrician Dr. Pierre Nobécourt died on Nov. 19, 1943.

CORRECTION

The pediatricians listed in the December issue (AM. J. DIS. CHILD. **63**:429, 1944) were certified by the American Board of Pediatrics in St. Louis, not in St. Louis and New York.

Abstracts from Current Literature

Metabolism; Infant Feeding Milk and Other Foods

THE RELATION OF URIC ACID EXCRETION TO BLOOD LACTIC ACID IN MAN. STANLEY T. MICHAEL, Am. J. Physiol. **141**:71 (May) 1944.

The author investigated the relation of the blood levels of lactate and pyruvate to the excretion of uric acid by the kidney after ingestion of lactic acid and compared the results with the blood lactate observed after the ingestion of sodium benzoate.

The excretion of uric acid was correlated to the lactic acid level in the blood for four hours after ingestion of lactic acid, the correlation coefficient being -0.82 ± 0.051.

After ingestion of lactic acid the blood lactic acid increased to values considerably higher than was the case after ingestion of sodium benzoate, although the reverse was true for the retention of uric acid, which was higher after ingestion of sodium benzoate.

A moderate correlation of uric acid excretion with the blood lactate level was observed in 25 resting and fasting subjects.

The correlation between the blood lactic acid level and the excretion of uric acid is considered evidence of a controlling influence of some phase of lactic acid metabolism on excretion of uric acid.

NOURSE, Cleveland. [AUTHOR'S SUMMARY]

THE INFLUENCE OF THE PROTEIN CONTENT OF THE DIET ON FAT DIGESTIBILITY. RICHARD H. BARNES, MARGARET F. PRIMROSE and GEORGE O. BURR, J. Nutrition **27**:179 (Feb.) 1944.

Lower protein intake was found to be associated with a lower digestibility of fats, according to Richard H. Barnes and others in their comparative studies of digestibilities of fats in rats receiving diets containing approximately 14 or 30 per cent protein. Well absorbed fats, such as lard, were influenced only slightly, while more poorly digestible fats, such as lard blended with hydrogenated fat and butter fat, were more seriously affected.

FREDEEN, Kansas City, Mo.

DIETARY PROTEIN AND PHYSICAL FITNESS IN TEMPERATE AND HOT ENVIRONMENTS. G. C. PITTS, F. C. CONSOLAZIO and R. E. JOHNSON, J. Nutrition **27**:497 (June) 1944.

G. C. Pitts and others conclude that even though protein does have a high specific dynamic action, protein intake may vary widely—from 75 to 150 Gm. daily—without effect on performance of intermittent work in the heat.

FREDEEN, Kansas City, Mo.

STUDIES ON THE COMPARATIVE NUTRITIVE VALUE OF FATS. HARRY J. DEUEL JR., ELI MOVITT and LOIS F. HALLMAN, J. Nutrition **27**:509 (June) 1944.

Deuel Jr. and his associates conclude that if fat per se is required for fertility and pregnancy this requirement may be as well satisfied by the various vegetable fats as by butter. From the standpoint of lactation, the different fats are also equally effective.

FREDEEN, Kansas City, Mo.

A COMPARISON OF THE DIETS OF SCHOOL CHILDREN NEW YORK CITY IN 1917 AND 1942. CHARLOTTE EVANS and ROSE LUBSCHEZ, J. Pediat. **24**:518 (M[illegible] 1944.

It is the opinion of many observers that the pre[sent] generation of children is in better physical condi[tion] than was a comparable group twenty years ago. T[his] change has been attributed to emphasis on public he[alth] education, particularly with respect to diet and consumption of protective foods.

A series of records of all articles of food consu[med] by 531 school children on a particular day in Jan[uary] 1917 and similar records for 277 children attend[ing] the same school in December 1942 were made avail[able] for analysis. The possible effects of a wartime econo[my] were comparable in these two years. The method u[sed] for collecting the data was the same in both years, a[nd] the records could be assumed to be representative of [the] dietary habits of the school population conside[red]. There was no significant difference in the age consti[tu]tion of the two groups, and there were equal numb[ers] of boys and girls in both groups. The neighborh[ood] remained the same and was representative of the w[hite] collar class in New York, with an average income [of] about $2,000 per year per family.

A random sample of 100 records of children betw[een] the ages of 10 and 12 years was selected from e[ach] group for analysis. The percentage of children ha[ving] stated portions of the various foods was calculated. A rough estimate of the adequacy of the diets in 1917 a[nd] in 1942 respectively was obtained by calculating [the] average number of servings of the various foods p[er] child per day and using these values to obtain [the] average protein, fat and carbohydrate content of [the] diets, the caloric intake and the vitamin content. A shortened method was adopted for obtaining these [meas]ures, and the calculations were based on published tab[les] of food values in which suitable allowance had b[een] made for losses due to cooking. The average val[ues] were compared with the recommended daily allowan[ces] of the various food constituents proposed by the N[a]tional Research Council Committee on Nutrition (19[44]).

Within the limitations inherent in dietary histories a definite improvement in the dietary habits of sch[ool] children of the present generation was observed. T[he] increased consumption of milk, citrus fruits and veg[e]tables is noteworthy, although it cannot be conclu[ded] that the dietary histories for 1942 reflect an optim[um] daily diet. The majority of the children in both ye[ars] had inadequate breakfasts and frequently an inadequ[ate] lunch. The vitamin content of the 1942 diet appe[ars] to be more adequate than that of the 1917 diet, with [the] exception of vitamin C, in which both diets were pr[ob]ably deficient. The consumption of protective foo[ds], especially foods containing vitamin C, was probab[ly] inadequate in both years. Further emphasis is nec[es]sary, particularly on the comparative nutritive values [of] different foods and the preferred methods for prepar[ing] cooked foods.

LUBSCHEZ, New York

Vitamins; Avitaminoses

AMINS AND HORMONES IN NUTRITION. BENJAMIN . SIEVE, Am. J. Digest. Dis. **11**:179 (June) 1944.

his very sensible article illustrates the association ndocrine imbalance and avitaminosis. Cases are

e author discusses the influence on vitamin ab- ion of such contributing factors as hormone dys- as, infection, gastrointestinal disease, reduced ry intake, emotional upset and trauma.

careful examination, a complete history and a er evaluation of apparently insignificant symptoms, routine laboratory tests, are essential for correct nosis.

e therapeutic recommendations consist of clearing oci of infection and contributing factors, restoring ndocrine balance by substitute therapy and ad- stering vitamins. Synergistic action of vitamins ndocrine hormones is indicated in this treatment.

MORRISON, Savannah, Ga.

EFFECT OF VITAMIN B COMPLEX DEFICIENCY ON HE WATER CONTENT OF THE BODY AND VARIOUS RGANS OF THE ALBINO RAT. JOHN HALDI, GLEN- ILLE GIDDINGS and WINFREY WYNN, Am. J. Physiol. **41**:83 (March) 1944.

he water content of the body of the albino rat was directly affected by depriving the animal of the min B complex.

NOURSE, Cleveland.

ECT OF SODIUM AND POTASSIUM CHLORIDE ON THE RENAL CLEARANCE OF ASCORBIC ACID. EWALD E. SELKURT and C. RILEY HOUCK, Am. J. Physiol. **141**:423 (May) 1944.

his study deals with the effects of infusion of ertonic sodium and potassium chloride solutions on reabsorption of ascorbic acid by the renal tubules. s were employed for the experiment. It was found the reabsorptive Tm (maximal rate) is significantly essed by these salts. This shows that they block ascorbic acid reabsorptive mechanism in some man- the nature of which is unknown.

STOESSER, Minneapolis.

AMIN B_1 HYPERSENSITIVITY WITH DESENSITIZATION. MOISES M. MITRANI, J. Allergy **15**:150 (March) 1944.

patient with hypersensitivity to vitamin B_1 was nsitized with subcutaneous injections of thiamine rochloride.

HOYER, Cincinnati.

AMIN A STORAGE AND FACTORS THAT AFFECT THE LIVER. C. C. CLAYTON and C. A. BAUMANN, J. Nutrition **27**:155 (Feb.) 1944.

. C. Clayton and C. A. Baumann found that the atic storage of vitamin A in the rat was relatively pendent of other biochemical processes taking place he liver. It was unaltered by the most potent car- ogenic agent known for the liver of the rat; it was ffected by factors that either depressed or enhanced formation of prothrombin, and it remained essen- ly the same whether the diets fed were high or low fat. These results suggest that vitamin A is stored the liver and released therefrom by a mechanism that is comparatively independent of other processes taking place in this organ.

FREDEEN, Kansas City, Mo.

PHYSIOLOGICAL AND BIOCHEMICAL FUNCTIONS IN NORMAL YOUNG MEN ON A DIET RESTRICTED IN RIBOFLAVIN. ANCEL KEYS, AUSTIN F. HENSCHEL, OLAF MICKELSEN, JOSEF M. BROZEK and J. H. CRAWFORD, J. Nutrition **27**:165 (Feb.) 1944.

Ancel Keys and others conclude that normal young men suffered no physiologic or clinical handicap by restriction to an intake of riboflavin of 0.31 mg. per thousand calories for a period of five months.

FREDEEN, Kansas City, Mo.

VITAMIN INTERRELATIONSHIPS. BARNETT SURE, J. Nutrition **27**:447 (June) 1944.

In chronic thiamine deficiency of the rat, uncomplicated by body tissue catabolism, Barnett Sure found great losses of riboflavin in the urine. He concludes that a definite thiamine-riboflavin interrelationship exists in chronic as well as in acute thiamine deficiency.

FREDEEN, Kansas City, Mo.

THE EFFECT OF VITAMIN E ON REPRODUCTION IN DOGS ON MILK DIETS. C. A. ELVEHJEM, J. E. GONCE JR. and S. W. NEWELL, J. Pediat. **24**:436 (April) 1944.

Although evaporated milk supplemented with iron, copper, manganese and cod liver oil supported normal growth in dogs, the pups of bitches fed this diet died within a few days after birth. The pups had muscular dystrophy or a hemorrhagic condition of the pericardium, lungs and brain. The results were not altered by the addition of α-tocopherol in the amount of from 5 to 25 mg. per week, vitamin K or thiamine to the diet of mineralized evaporated milk given during the period of gestation. However, when the amount of α-tocopherol was raised to 40 mg. per week normal reproduction occurred, although 1 of the 4 pups showed slight muscular dystrophy. Since the limiting effect of evaporated milk became evident only in the offspring, it is doubtful that this observation can have any extensive application in practical nutrition. As more is learned about vitamin E in the nutrition of infants it may be important to recognize that evaporation tends to reduce the vitamin E content of milk.

GONCE JR., Madison.

Prematurity and Congenital Deformities

CONGENITAL MALFORMATIONS INDUCED IN RATS BY MATERNAL NUTRITIONAL DEFICIENCY. JOSEF WARKANY and ELIZABETH SCHRAFFENBERGER, J. Nutrition **27**:477 (June) 1944.

Warkany and Schraffenberger in their studies with rats conclude that the abnormalities were caused by a prenatal deficiency of riboflavin.

FREDEEN, Kansas City, Mo.

HEREDITARY CLEIDOCRANIAL DYSOSTOSIS. D. D. SALMON, Radiology **42**:391 (April) 1944.

A soldier of French-Irish descent, 21 years of age, had experienced extreme fatigue and a feeling of weight in the neck and shoulders after calisthenics. An examination revealed a "soft spot" in the midline of the frontal and parietal bones, ability to approximate his

shoulders anteriorly and absence of collar bones. His deciduous teeth were lost late in adolescence and were never replaced by permanent teeth. The patient was short and stocky. A family history suggested that his father and a sister had some of these unusual features.

Roentgenograms revealed partial aplasia of the clavicles, absence of osseous tissue in the frontal bone in the midline, open skull sutures, wormian bones, no development of frontal sinuses, underdevelopment of the maxilla, irregularities of the teeth and nonpneumonization of the mastoids.

The author calls attention to the preosseous and predentinal origin of this hereditary condition as emphasized previously by Anspach and Heupel.

ANSPACH, Chicago.

Acute Contagious Diseases

KELOIDS AS SEQUEL TO VARICELLA. EUGENE F. TRAUB, Arch. Dermat. & Syph. **49**:278 (April) 1944.

The author reported that a 6 year old boy who had previously shown no tendency to the formation of keloids promptly acquired more than a dozen after an attack of chickenpox.

McINTOSH, Iowa City.

VARICELLA VESICLE IN EXTERNAL AUDITORY CANAL. B. POLAND, Arch. Otolaryng. **37**:852 (June) 1943.

Reported here is a case of a varicella vesicle in the external auditory canal. The purpose of the report is to point out the possibility of involvement of the ear by lesions of chickenpox and to stress the necessity for thinking of this possibility in the differential diagnosis of otitis media complicating chickenpox.

GREENWOOD, Chicago.

OUTBREAK OF SEPTIC SORE THROAT AND SCARLET FEVER IN A SCHOOL. Health News **20**:87 (May 24) 1943.

Fifty-five students from 5 to 17 years of age in an upstate New York school had septic sore throat and scarlet fever. No fatalities were reported. The epidemic was traced to tapioca pudding which had stood overnight without refrigeration. The pudding had been prepared by a woman ill with "sore throat."

AIKMAN, Rochester, N. Y.

GROUP B STREPTOCOCCI AND MALIGNANT DIPHTHERIA. ELAINE UPDYKE and MARTIN FROBISHER JR., J. Bact. **47**:474 (May) 1944.

From time to time there have been reports that in malignant diphtheria certain streptococci and diphtheria bacilli act synergistically. The authors isolated a beta type hemolytic streptococcus of Lancefield's group B from persons with malignant diphtheria in Baltimore along with mitis-like diphtheria bacilli. They found that streptococci of other groups were frequently present in diphtheria but did not seem to be regularly associated with malignancy. The synergistic action of some of the group B strains of streptococci was investigated. Very careful experiments were performed, and it appears clear from them that there is a definite synergistic action between certain streptococci and diphtheria bacilli. For details concerning the experiments the original report should be consulted.

STOESSER, Minneapolis.

HYPERIMMUNE WHOOPING COUGH SERUM. AN... McGUINESS, JANET G. ARMSTRONG and HARRI... FELTON, J. Pediat. **24**:249 (March) 1944.

Hyperimmune whooping cough serum obtained ... donors immunized with phase I pertussis vaccine ... employed in passive immunization of 308 infants ... children subsequent to exposure to whooping cough ... in the treatment of 442 patients with whooping co... after the onset of paroxysms.

The results obtained closely parallel those report... earlier studies, which indicated that the serum wa... considerable value in the prevention and treatme... the disease.

The possible use of toxoid and/or purified a... tinogen, either alone or in combination with vaccine... the development of hyperimmune serum is in the pr... of investigation.

[AUTHORS' SUMMAR...

IMMUNIZATION WITH COMBINED DIPHTHERIA AND T... ANUS TOXOIDS (ALUMINUM HYDROXIDE ADSO... CONTAINING HEMOPHILUS PERTUSSIS VACCINE. ... MILLER JR., JEANETTE B. HUMBER and JAME... DOWRIE, J. Pediat. **24**:281 (March) 1944.

One hundred and seventy-two children at the C... dren's Clinic of Stanford University Medical Sc... were given two injections with an interval of tw... weeks, of 1 cc. of a mixture of aluminum hydroxi... adsorbed diphtheria and tetanus toxoids conta... phase I Haemophilus pertussis vaccine (20 billion ... ganisms per cubic centimeter). The titers of tetanus a... toxin and of H. pertussis agglutinins in the recipi... blood were studied at suitable intervals for 149 and ... subjects respectively, and Schick tests were perfor... on 114 children. Exceptionally high and enduring le... of tetanus antitoxin were obtained, and all the Sc... tests were negative. The response to pertussis vacc... tion was not entirely satisfactory, but it is believed ... this fault can be corrected by giving an extra dos... pertussis vaccine midway between the two injections ... combined toxoid and vaccine. Aluminum hydroxi... adsorbed toxoid appears to be superior in antigeni... to alum-precipitated toxoid, particularly with respe... tetanus. No abscesses were observed. One hundred ... seventeen of the children were under 1 year of age, ... all but 8 of the remainder were under 6 years.

FABER, San Francisco.

RIBOFLAVIN, PANTOTHENIC ACID AND BIOTIN EXCRE... TESTS IN PATIENTS WITH PARALYTIC POLIOMYELI... W. O. FROHRING and JOHN A. TOOMEY, J. Ped... **24**:293 (March) 1944.

The authors measured the urinary excretion of ri... flavin, pantothenic acid and biotin by patients w... paralytic poliomyelitis.

The values for pantothenic acid were within nor... limits. In 8 of 10 specimens of urine examined ... amount of riboflavin was higher than that found ... Strong, Feeney and Moore in the urine of normal p... sons. The content of biotin seemed high, but infor... tion about normal values was lacking, and no conc... sions were drawn.

TOOMEY, Cleveland.

ACUTE SEROUS MENINGITIS IN SCARLET FEVER. LE... K. SWEET and MARK H. LEPPER, J. Pediat. **24**:... (March) 1944.

This paper describes a syndrome of acute serous m... ingitis occurring in 11 of 1,503 patients with sca... fever treated in the Gallinger Municipal Hospital. ...

al and laboratory findings were uniform. There no apparent relationship to age, sex or race. The ition occurred most frequently in the spring and

Ten of the patients had moderately or severely scarlet fever; 1 had mild scarlet fever. There was niform therapy prior to the onset of the complication. he usual course included an initial good response he scarlet fever, with abatement of the fever and toxic symptoms. Between the fifth and the eighth after the onset of scarlet fever there was an exbation of fever and toxemia, usually with vomiting dehydration. The leukocyte count varied from 00 to 32,000 per cubic millimeter of blood with an ease of immature neutrophils. In no instance was e a septic focus to which the fever could be due. hortly after the secondary rise in temperature signs meningeal irritation developed in all the patients. cerebrospinal fluid showed pleocytosis; the number ells per cubic millimeter ranged from 32 to 1,100, all patients except 2 having over 100 cells. Usually er cent or more of the cells in the spinal fluid were phocytes. The amount of dextrose in the spinal fluid normal; the amount of protein was increased to mg. per hundred cubic centimeters, and the culture sterile. The cerebrospinal fluid became normal t to twenty-eight days after the secondary fever. secondary fever itself subsided after periods varyfrom five to nine days, or eleven to sixteen days r the initial onset of the scarlet fever. No sequelae he disease were encountered.

pecimens of cerebrospinal fluid from 9 patients were igerated and sent directly to the National Institute Health, Bethesda, Md., where they were injected iously into white mice, white rats, cotton rats, hamrs, guinea pigs, rabbits and a monkey. No virus obtained from the spinal fluid of any patient. The od of 2 patients, tested after eighteen and twentyr months respectively, failed to neutralize the virus ymphocytic choriomeningitis.

he cause of the complication is uncertain. The ical features strongly suggest that it is caused by iltrable virus, but this impression is at present unfirmed.

Detailed case reports of the 11 patients are presented.

SWEET, Washington.

HOOPING COUGH CONTROL. JOSEPH H. LAPIN, J. Pediat. **24**:331 (March) 1944.

The control of whooping cough through a public lth program is both necessary and practicable at present time. Health departments should establish ilities for routine prophylaxis for well children, for king cough plates and nasopharyngeal and throat abs for diagnosis and for serum prophylaxis for ldren who have been exposed to infection.

[AUTHOR'S SUMMARY.]

EATMENT OF MENINGOCOCCIC MENINGITIS AND SEPTICEMIA. C. W. CORY, C. E. ABBOTT JR. and E. G. TRUSZKOWSKI, J. Pediat. **25**:35 (July) 1944.

An analysis was made of the cases of 148 patients h meningococcic meningitis and meningococcemia ated at the station hospital, Jefferson Barracks, Mo., ring the period from June 1941 through April 1943. e first 48 patients were treated with sulfanilamide, ich was supplemented by antimeningococcus serum 83.3 per cent of this number. The later group of patients was treated with sulfadiazine and with its dium salt when indicated, and antimeningococcus rum was given to only 14 per cent of them.

All the symptoms observed which are referable to the disease are described. The most constant were severe headache, stiff neck, sore throat and petechial rash.

Of the neurologic findings, the high incidence of Kernig's sign and of Brudzinski's neck sign is noteworthy. By comparison, the number of patients exhibiting Babinski's sign was small.

Initial lumbar punctures were performed on all patients on their admission and were repeated only when severe headache persisted or when continued increasing intracranial pressure was evident.

PREVALENCE OF POLIOMYELITIS IN THE UNITED STATES IN 1943. C. C. DAUER, Pub. Health Rep. **59**:712 (June 2) 1944.

In 1943 forty-eight states reported 12,429 cases of poliomyelitis, which is the largest number since records have been kept except in 1916 and 1931; in these years there were respectively 27,363 and 15,790 cases. The disease was widespread along the Pacific coast and in the Southwest. In the East the rate was low. The city having the highest morbidity rate was Pueblo, Colo., with 99.6 cases per hundred thousand inhabitants. Chicago reported the greatest number of cases—1,053—but the morbidity rate was only 31 per hundred thousand.

There has been no consistent interval of time between severe outbreaks. Nor do differences in geography, climate, size of state, distribution, density or racial characteristics enter as factors. A period of about three decades does not seem to be sufficiently long to permit any definite conclusions regarding trends in incidence.

SANFORD, Chicago.

REVACCINATION AS A MEASURE OF IMMUNITY TO SMALLPOX. E. S. HORGAN and MANSOUR ALI HASEEB, J. Hyg. **43**:337 (April) 1944.

Serologic methods for the determination of smallpox immunity are considered unlikely to provide a diagnostic method of sufficient accuracy. In revaccinations for success greater emphasis must be put on the skill of the operator, the potency of the lymph and the number of insertions (scratches) than in primary vaccination.

Experimental work done on 107 previously vaccinated recruits showed 12 primary takes, 48 vaccinoid reactions and 28 immune reactions. Study of materials of different strengths which were used showed little advantage in the stronger (elementary body suspension of high potency) over the standard vaccine lymph, but the value of two insertions (scratches) over one was very marked and is recommended over the one scratch method. Otherwise, some vaccinoid reactions will be reported as immune reactions.

No mention is made of the multiple puncture method of vaccination.

JANNEY, Wauwatosa, Wis.

DIPHTHERIA IN GLASGOW 1934-42. H. S. CARTER, J. Hyg. **43**:341 (April) 1944.

During the period from 1934 to 1942, 11,312 cases of diphtheria were studied. The case fatality rate declined during the period, so that the 1942 rate was one third of that of 1934, but the strain of the Corynebacterium diphtheriae has shown a marked change, with a reduction in the intermedius and mitis forms and an increase in the gravis form to about double the numbers of the first two combined.

The case mortality, as expected, was highest in the age group 0 to 4 years—8.3 per cent gravis, 1.3 per cent intermedius and 5.4 per cent mitis, or 5.8 per cent; while that of the age group 15 and older was 0.7 per cent.

The nonimmunized child in Glasgow is four and one-half times more liable to contract diphtheria than the immunized child, and the risk of a fatal issue among the immunized is slight.

JANNEY, Wauwatosa, Wis.

A SIMPLE MEDIUM FOR THE DETECTION OF CORYNEBACTERIUM DIPHTHERIAE. P. M. ANDERSON, M. J. Australia **1**:213 (March 11), 1944.

A simple tellurite medium (potassium tellurite, glycerolated sheep's blood and nutrient agar) is described, which is highly selective for Corynebacterium diphtheriae and which, in addition, permits a confirmatory microscopic examination to be carried out. The medium does not, however, permit the prompt classification of the colonies into the gravis, mitis and intermedius types.

GONCE, Madison, Wis.

Acute Infectious Diseases

HYPOGLYCEMIC REACTION WITH CONVULSIONS IN ASCARIASIS (CASE REPORT). LUDWIG L. FRANK, Am. J. Digest. Dis. **11**:195 (June) 1944.

This report deals with the case of a 9 year old boy who had convulsions with fever at intervals from the age of 8 months. The only significant finding was the passage of roundworms (Ascaris lumbricoides). The fasting blood sugar was 85 mg. per hundred cubic centimeters in the morning and 50 mg. per hundred cubic centimeters before noon. The boy passed six ascarids, and no further convulsions occurred during the following eleven months.

The author postulates that the triad of ascariasis, convulsions and hypoglycemia may be a clinical entity. Certain hypotheses are given.

This case is inconclusive in that the blood sugar value of 50 mg. per hundred cubic centimeters does not usually produce symptoms of hypoglycemia [abstracter].

MORRISON, Savannah, Ga.

TULAREMIA IN INFANCY. W. A. DANIEL JR., J. Pediat. **24**:326 (March) 1944.

Although tularemia in infants and children has been infrequently reported, the disease is apparently more common than suspected. The saliva of dogs and cats has been found to harbor the organisms frequently.

Tularemia in a 22 month old white boy is reported. The onset was moderately sudden with a high fever and convulsions. Physical examination revealed no significant signs other than a thin, watery nasal discharge and moderate injection of the pharynx. There were 3,080,000 red cells per cubic millimeter of blood, 11 Gm. of hemoglobin per hundred cubic centimeters and 26,800 white cells per cubic millimeter, of which 77 per cent were polymorphonuclear neutrophils. Roentgen examination of the chest revealed no pulmonary or cardiac abnormalities. The diagnosis was made by means of agglutination studies of the blood. The course of the disease was characterized by an acute phase with high fever and a period of gradually decreasing temperature. The child did not appear to be ill except when the fever was at its height. Treatment was with sulfadiazine and antitularemia serum. Neither therapy appeared to influence the course of the illness. The acute phase lasted approximately one month, and clinical well-being apparent two weeks later.

It is believed that the antitularemia serum was g in too small a quantity. Tularemia remains a dis especially difficult to diagnose in children, a dis without truly adequate therapy, but fortunately a dis of low mortality.

An extensive bibliography on tularemia in infants children is appended.

DANIEL, Montgomery, Al

RATBITE FEVER CAUSED BY STREPTOBACILLUS MON FORMIS. EDGAR ROSEN and BERNARD S. DENZ J. Pediat. **24**:544 (May) 1944.

Fever following a rat bite may be caused by eit of two etiologic agents, Spirillum minus and Strep bacillus moniliformis (also commonly known as Hav hillia multiformis). These two organisms, biologic different, produce similar clinical pictures. The dise caused by Spirillum minus is produced only by the b of a rat or another animal carrying the organism. T Japanese speak of this disease as sodoku. Streptobacil moniliformis septicemia is caused by a rat bite and a follows ingestion of contaminated food. The latter d ease has been called Haverhill fever.

Spirillum minus was isolated from a case of rat b fever in 1917. Three years previously Schottmuel isolated by blood culture an organism which he term Streptothrix muris ratti. The existence of this organi has been confirmed by many authors, and the name n applied is Streptobacillus moniliformis. In 1926, in t town of Haverhill, Mass., an identical organism w isolated during a small epidemic which was supposed caused by a contaminated milk supply. This organi was called Haverhilla multiformis.

The discovery of Streptobacillus moniliformis both cases of fever due to a rat bite and in cases of syn toms apparently due to ingestion of contaminated fo has caused considerable confusion. Aside from the loc lesion the symptoms are the same. Both conditio should be thought of as Streptobacillus monilifor septicemia with different portals of entry of the infec ing agent.

The disease is characterized by an abrupt onset, wi chills, fever, vomiting and headache, usually followe an incubation period of about three to six days, althou sometimes the incubation period is as long as for sodok the spirillary form of the disease. Until recently the has been no specific treatment of Streptobacillus moni formis disease.

DENZER, New York.

THE SIGNIFICANCE OF LESS COMMON SHIGEL STRAINS IN INSTITUTES FOR MENTALLY DEFICIE CHILDREN. OSCAR FELSENFELD and VIOLA M YOUNG, J. Pediat. **25**:56 (July) 1944.

Three hundred and seventy-two strains of Shige were isolated from 1,213 mentally deficient childre and 4,584 mentally ill adults. The percentage of carri found was 14.4 for the newly admitted children, 2 for the chronically institutionalized children, 0.3 for t newly admitted adults and 3.4 for the adults who ha been confined to state hospitals for a long perio Twelve and three-tenths per cent of the children a 5.9 per cent of the adults who excreted these organis were found to be harboring simultaneously two or thr types of Shigella.

The type of Shigella most frequently found in childre was S. ambigua, which was followed in order of fr quency by S. sonnei, S. alkalescens and S. paradys teriae Z, 103, V and 88; the Shigellae isolated from adult presented in the same order, were S. sonnei, S. alkal

, S. paradysenteriae 103, S. ambigua and S. para-enteriae Z, V and 88. The frequent appearance of S. igua in children may be explained by an outbreak ysentery caused by this organism in a children's pital. Members of the S. dispar subgroup, i. e., S. ampensis and S. ceylonensis, were found in 2.8 per of all the children examined and in 0.2 per cent he adults. As these organisms were rarely encoun-d in diarrheic stools in which no other organisms known pathogenicity were present, the authors con-e that S. dispar plays but a secondary role in caus-enteric infections. S. gintottensis and equirulis-like anisms found in the course of this study are not sidered pathogenic. No conclusions could be drawn cerning the significance of S. rettgeri, which was vered from stools of 2 patients. This organism, vever, should not be classified in the genus Shigella. actose-fermenting Shigellae failed to produce aggluns to a significant level in the blood of carriers of or three strains. Other types of Shigella, however, e satisfactory serologic tests when experiments in lutination with alcohol-treated antigens and with namide extracts were performed.

AUTHORS' SUMMARY.

OKWORM INFESTATION. T. E. LOWE and H. O. LANCASTER, M. J. Australia **1**:289 (April 1) 1944.

The authors studied 386 cases of recent mild hook-rm infection occurring among soldiers with malaria. nically, the infection produced cutaneous lesions, dys-sia and pronounced eosinophilia but no anemia ater than that common to malarial patients. The nber of eosinophilic cells varied between 500 and 0 per cubic millimeter in 70 per cent of the patients, compared with less than 400 per cubic millimeter in per cent of patients convalescent from malaria who e free from helminth infection. A considerable fall the number of eosinophils preceded a malarial rigor. comparison between the effectiveness of various ant-minthic drugs indicated that emulsions containing m chenopodii with either carbon tetrachloride or achlorethylene are the most effective.

GONCE JR., Madison.

MPOSIUM ON TROPICAL DISEASES. BOYD READING, RAFAEL DE LA PORTILLA and FELIX HURTADO, Bol. Soc. cubana **15**:533 (Aug.) 1943.

This is a translation by Dr. Felix Hurtado and Prof. fael de la Portilla of the round table discussion on pical diseases of the American Academy of Pediat-s, which was reported in the *Journal of Pediatrics* :612-633 [May] 1933). There are four pages of nments in English by Dr. F. Hurtado on the dis-es discussed. He emphasizes the part played by Carlos Finlay of Cuba in the study of yellow fever its transmission and states that yellow fever and gue are not now known in Cuba. He points out the portance of air travel in the possible spread of yellow er and of cholera.

KEITH, Rochester, Minn.

Diseases of Blood, Heart and Blood Vessels and Spleen

EMANN-PICK DISEASE. MARK F. CANMANN, J. Pediat. **24**:335 (March) 1944.

The literature on Niemann-Pick disease is reviewed, a new case is reported.

The patient was a 10 week old girl of Italian and English-Irish parentage. Her symptoms started at birth and terminated seventy-three days later. An autopsy was performed on the trunk, and the conditions observed are reported in this paper. Dr. Smith Freeman, of Northwestern University, analyzed formaldehyde-fixed liver, and his results expressed in terms of dry liver (by weight) were as follows: total fat, 10 Gm. per hundred grams; total cholesterol, 0.818 Gm. per hundred grams, 8.2 per cent of the total fat, and lipoid phosphorus expressed as lecithin, 3.78 Gm. per hundred grams, or 38 per cent of the total fat. The normal range according to Ralli, Rubin and Rinzler is essentially the same.

With this report, 60 proved cases of Niemann-Pick disease could be found in the literature, in 32 of which the patients were female and in 26 male. Thirty of the 60 persons with the disease were Jewish, and 32 per cent had a familial history of the disease.

[AUTHOR'S SUMMARY.]

Diseases of Nose, Throat and Ear

OBSERVATIONS ON THE EPIDEMIOLOGY OF STREPTOCOCCAL PHARYNGITIS AND THE RELATION OF STREPTOCOCCAL CARRIERS TO THE OCCURRENCE OF OUTBREAKS. ANN G. KUTTNER and ELMA KRUMWIEDE, J. Clin. Investigation **23**:139 (March) 1944.

During a six year period of study of major and minor outbreaks of streptococcic pharyngitis in groups of rheumatic children in a sanatorium, (*a*) major outbreaks were due to group A streptococci of a single type not previously present and were not preceded by a rise in carrier rate; (*b*) minor outbreaks were preceded by a slow spread of the organisms from carriers to other persons without infection at first; (*c*) sporadic infections were contracted directly from carriers and were not preceded by a dissemination of the streptococci to healthy persons.

Observations on the carrier state: (*a*) Twenty-nine per cent of the children admitted during the summer and fall months were carriers of group A hemolytic streptococci, but with few exceptions these microorganisms did not spread to other persons and disappeared after a few months; (*b*) the epidemic-inducing types of streptococci persisted longer in "postinfection" than in "contact" carriers; (*c*) the length of the carrier state was not related to the presence or absence of tonsils.

GUEST, Cincinnati.

EXPERIMENTS IN THE SYNTHESIS OF CLINICAL METHODS INTO A PROGRAM OF REHABILITATION. OLLIE L. BACKUS and M. HARRIET M. DUNN, J. Speech Disorders **9**:1 (March) 1944.

Two summers of corrective work at Pennsylvania State College and at the University of Michigan's speech clinics were provided for 28 persons with speech defects, including patients with cleft palate, deafness, articulatory difficulty and spasticity. The procedure was a mixture of group and individual instruction toward rehabilitation. Each patient was in the clinic during a morning period. The groups were divided according to the ages of the subjects. There were periods for relaxation, blowing exercises, discussion and other activities. The authors claim that the group made greater progress than did persons who received only individual instruction. The control subjects are not described in the article.

PALMER, Wichita, Kan.

AUDITORY MEMORY SPAN FOR SPEECH SOUNDS: NORMS FOR CHILDREN. RUTH WATT METRAUX, J. Speech Disorders **9**:31 (March) 1944.

Four hundred and fourteen children, 199 boys and 215 girls, ranging in age from 4 years and 6 months to 12 years and 5 months, were tested with a modification of Anderson's scale of auditory memory span. There are significant differences between various age groups for memory of both vowels and consonants. The difference between adjacent age levels is not always statistically significant. There is no sex difference for auditory memory span. There appears to be no significant correlation between mental age and auditory memory span. Since it does not seem to matter what part of the test is given first, fatigue and lowered attention do not contribute significantly to relatively lower scores on the test on consonants.

PALMER, Wichita, Kan.

Diseases of the Gastrointestinal Tract, Liver and Peritoneum

ON THE MECHANISM OF THE ADAPTATION OF PANCREATIC ENZYMES TO DIETARY COMPOSITION. M. I. GROSSMAN, HARRY GREENGARD and A. C. IVY, Am. J. Physiol. **141**:38 (March) 1944.

Substitution of dextrose for corn starch in a balanced diet results in increased amylase content of the pancreatic tissue. Thus in the case of starch a humoral agency involving dextrose seems to regulate the amount of amylase produced. On the other hand, the substitution of casein hydrolysate for casein in a balanced diet produces a depression of trypsin content in the pancreatic tissue. This suggests a nervous reflex factor or a more complex hydrolytic product of protein, such as proteose, as the stimulus.

NOURSE, Cleveland.

HISTAMINE IN HUMAN GASTRIC MUCOSA. BENEDICT TROCH, CHARLES F. CODE and OWEN H. WAGENSTEEN, Am. J. Physiol. **141**:78 (March) 1944.

The histamine activity extractable from human gestric mucosa was studied in 10 cases. In 9 cases extracts were made of the fundic mucosa, and these were found to contain activity equivalent to 3.5 to 24.1 mg. of histamine per kilogram of fundic mucosa, with an average value of 10.2 mg. of histamine per kilogram of mucosa. In 4 cases extracts were made of antral mucosa. The histamine activity of these extracts ranged from 3.0 to 12.5 mg. of histamine per kilogram of mucosa, with an average value of 5.8 mg. of histamine per kilogram of mucosa. In 3 cases the histamine activity of the fundic mucosa was compared with that of the antral mucosa. In 1 case the antral mucosa contained much less activity than the fundic mucosa, but in the other 2 cases the concentration was approximately the same. On subcutaneous injection into dogs with Heidenhain pouches all the extracts produced a prompt secretion of acid gastric juice. The possible significance of some of the limitations of these results are discussed.

NOURSE, Cleveland.

CONGENITAL PYLORIC STENOSIS. CHARLES A. VANCE, Ann. Surg. **119**:351 (March) 1944.

Congenital pyloric stenosis, better termed infantile pyloric stenosis, is the most common condition that requires surgical treatment in the first few weeks of life. The mortality prior to 1908 was 50 to 75 per cent; at the present time it has been reduced to 1 to 2 per cent.

The author gives an interesting history of early reported cases and of the development of treatment from gastroenterostomy to Ramstedt's operation (pyl myotomy).

The cause of congenital pyloric stenosis is not u stood. The condition appears in male children dominantly, in a ratio of about nine or ten to There seems to be a preponderance of first-born among those affected. If the five cardinal symptom present, namely, scanty stools, projectile vomiting of weight and dehydration, visible peristalsis and pable tumor, roentgenologic examination is not nece nor desirable.

The author prefers ether anesthesia and stresses importance of preoperative and postoperative care.

BANTIN, Oma

CONGENITAL ATRESIA OF THE ESOPHAGUS W TRACHEO-ESOPHAGEAL FISTULA. ALBERT O. STE TON and MAYNARD D. KNIGHT, Ann. Surg. **11** (April) 1944.

The symptoms are constant and diagnostic. The fant drools excessive amounts of mucus and saliva is subject to attacks of choking and cyanosis i taking of fluids by mouth. If a soft rubber cathe inserted down the esophagus it reaches an obstru 10 to 12 cm. from the anterior alveolar margin. A ciated anomalies are found in about one half o recorded cases.

The history of congenital esophageal atresia tracheoesophageal fistula is one of long and rep failures in efforts which have been made to treat condition. The authors favor the transpleural appro with ligation of the fistula and a primary anasto if technically possible. Four cases are reported.

BANTIN, Oma

ANNULAR PANCREAS PRODUCING DUODENAL OBST TION. ROBERT E. GROSS and TAGUE C. CHRISM Ann. Surg. **119**:759 (May) 1944.

About 50 cases of annular pancreas in man have b reported. In 39 the anomaly was found incidentall autopsy, and there were few or no symptoms. duodenal obstruction may be complete, so that ther evidence of obstruction in the first few days of life it may be so slight that symptoms do not appear adult life.

The symptoms are those of duodenal obstruction: gastric pain, nausea, vomiting and possibly malnutri Serial roentgenologic examination with the aid of bar sulfate should give rather conclusive proof of a par block. In occasional cases the symptoms and phys findings are those of an acute pancreatitis.

If symptoms of duodenal obstruction appear, should be relieved by surgical intervention. The auth report the case of a newborn infant with duodenal struction due to an annular pancreas, on whom operation was successfully performed on the third of life.

BANTIN, Omah

THE RELATION OF PANCREATIC ACHYLIA TO MECONI ILEUS. SIDNEY FARBER, J. Pediat. **24**:387 (Ap 1944.

From the postmortem examination of 18 patients w meconium ileus support is obtained for the sugges originally made by Landsteiner, that meconium ileus be caused by an obstructive lesion of the pancreas. P creatic achylia, which may be anticipated from the tologic picture of the pancreas, was proved by meas ment of the tryptic activity of the duodenal drainage

ing and 1 recently dead patient with meconium There was no evidence of tryptic activity in the enal drainage of either patient. Experiments per- ed both in vitro and in vivo with the altered nium found in patients with meconium ileus have n that this material may be brought into a semi- or fluid state by the action of dilute saline solu- of extracts of the pancreas.

he application of these observations to the treatment atients with meconium ileus has resulted in relief he intestinal obstruction. Caution in prognosis ld be exercised, however, for even when the intes- obstruction caused by altered meconium is relieved he solvent action of pancreatic extracts possible er may still exist because of the nutritional con- ences of pancreatic achylia or because the lesion of pancreas is but one of many similar lesions involv- a number of secreting structures in the body. Me- um ileus caused by pancreatic achylia should be erentiated from the clinically much less important issation of meconium that occurs in patients with ial or complete obstruction of the intestine in the natal period.

FARBER, Boston.

DY OF SERUM LIPIDS IN THE CELIAC SYNDROME. ARILD E. HANSEN and LUIGI LUZZATTI, J. Pediat. 24:417 (April) 1944.

The metabolism of fat of 10 children with the celiac drome who were under observation in the department pediatrics of the University of Minnesota during a iod of two years was studied. In 4 of the 6 patients o died, cystic fibrosis of the pancreas was demon- ted at autopsy, while the clinical picture and labo- ory data suggested the same diagnosis for the other Of the 4 children who were still living, 1 was con- ered to have cystic fibrosis of the pancreas, whereas other 3 were considerably improved and had left hospital.

Studies of the serum lipids and analyses of the fecal for the 10 patients revealed the following conditions: For 8 of the patients values for total fatty acids, tone-soluble fatty acids (cholesterol esters plus neu- fat), acetone-insoluble fatty acids (phospholipids), l cholesterol, free cholesterol and cholesterol esters re within the range of normal variability. 2. The ine numbers of the fatty acids in all fractions of the um lipids of these patients were abnormally low. After the administration of a fatty meal (cream) no rease in the serum lipids was noted in 5 of the sub- ts; in 1 patient a slight rise occurred, and in the er 2 the increase was normal. It was considered un- ely that these 2 patients had cystic fibrosis of the ncreas. 4. Administration of a meal containing fat a high degree of unsaturation (corn oil) revealed t no better absorption occurred than after ingestion fat with a low degree of unsaturation (cream).

As a result of the investigations the following con- sions were drawn by the authors:

1. These studies suggest that the human organism is able to synthesize fat of a high degree of unsaturation. 2. It appears that the determination of the iodine mbers of the serum fatty acids of patients with the liac syndrome may offer a means of ascertaining the ility of the subjects to absorb fat. The use of this latively simple procedure may be helpful in confirming e diagnosis and in evaluating clinical improvement, pecially in view of the fact that steatorrhea, which is dinarily present in patients with the celiac syndrome, often lacking because the patients are usually main- ined on a diet relatively low in fat.

3. The deficiency in the absorption of fat that is a part of the celiac syndrome is difficult to explain in the light of present knowledge of the physiology of metabolism of fat.

HANSEN, Galveston, Texas.

AN EPIDEMIC OF COMMON INFECTIOUS JAUNDICE. KENNETH W. MCLEOD, J. Pediat. 24:454 (April) 1944.

An epidemic of common infectious jaundice, involving 100 cases, which occurred in an institution for the feebleminded is reported.

The incidence of jaundice was noticeably higher in the idiot group than in the more active moron group. This fact was considered important with reference to the mode of transmission, suggesting that some route other than droplet infection was responsible. The younger age group was most commonly affected. The largest number of cases occurred during the fall and winter. The disease appeared to be communicable by contact. The incubation period was estimated to be thirty days.

The clinical characteristics of the disease were anorexia, malaise, jaundice, fever and clay-colored stools. Usually there was constipation. The abdomen was usually tender and the liver palpable. The illness lasted ten days to three weeks. No organisms could be demonstrated in biliary drainage or in cultures of biliary drainage or stools. Spirochetal jaundice (Weil's disease) did not occur concurrently. Hepatic function as measured by galactose tolerance tests showed impairment.

There is no specific treatment for common infectious jaundice. A diet high in carbohydrates and magnesium sulfate was the treatment commonly used, although a high fat diet and antispasmodic drugs (belladonna, phenobarbital) were tolerated equally well. There were no complications.

A review of the literature suggests that the disease is spread from person to person and that the contact is nearly always close. A possible relationship between injections of arsenicals and infectious jaundice has been considered by some authors. In the epidemic reported this factor was not significant.

All attempts to transmit the infecting agent to animals have been unsuccessful. Jaundice identical in nature with infectious jaundice occurred after the administration of serum as prophylactic immunization against measles, and the same sequence of events occurred after the use of yellow fever vaccine. The incubation period was long. The disease is usually mild, but it is occasionally severe in older adults and in pregnant women.

MCLEOD, Lapeer, Mich.

BEZOAR CAUSING INTESTINAL OBSTRUCTION. ROY P. FORBES, J. Pediat. 24:574 (May) 1944.

A case of a 13 year old girl with a hair ball as the cause of intermittent and finally fatal intestinal obstruction is presented in detail. Her past history revealed a diagnosis of acute appendicitis at 10 years, followed by surgical intervention without relief of symptoms. Vomiting to the point of emaciation occurred with spontaneous recovery, apparently as the bezoar was expelled from the pyloric ring back into the stomach, where it caused no further symptoms until three years later. Laboratory findings were of interest because of marked hypochloremia and low serum sodium. The blood pressure was 58 systolic and 18 diastolic. A tentative diagnosis of adrenal cortex insufficiency was made. Death after twenty-four hours of hospitalization

was followed by autopsy, which showed intestinal obstruction, with a bezoar in the ileum and a volvulus below the site. The adrenals were normal.

GENGENBACH, Denver.

DYSCHEZIA AND MEGACOLON. ARTHUR HURST, Radiology **42**:128 (Feb.) 1944.

Normal defecation depends on a conditioned reflex. An infant is trained to open his bowels when he is put on a chamber, no mental process being concerned in the act. In dyschezia the retention of solid feces in the rectum, which is normally empty except immediately before defecation, may give rise to a number of reflex symptoms, such as headache and general malaise, which disappear directly when the bowels are open. The instantaneous relief proves that these symptoms are not caused by autointoxication. Moreover, experimental distention of the rectum with a large plug of wool produces identical symptoms. Pressure on the surrounding parts by the retained feces also gives rise to perineal discomfort. Dyschezia can be cured by restoring to activity the defecation reflex, which has been allowed to become inefficient through neglect and through interference with its normal performance by habitual use of aperients.

Megacolon is a condition in which the rectum and the pelvic colon and sometimes the whole of the large intestine are dilated and hypertrophied, although no organic obstruction is present. The name Hirschsprung's disease, which is often used to describe megacolon in children, should be discarded. No clear dividing line can be drawn between the megacolon of children and that of adults. Megacolon in children is rare, the author having encountered only 11 cases, compared with 38 instances in which it occurred in adults. In children megacolon occurs almost exclusively in boys (10 out of 11 of the author's cases), whereas in adults the incidence is equal in the two sexes (20 males and 18 females in the author's series). It is probable, therefore, that in the majority of cases in which megacolon is observed in adults it does not date from childhood.

The author believes that the primary factor in the pathogenesis of megacolon in all cases is achalasia of the sphincter ani, absence of relaxation of the sphincter, which should occur when the rectum contracts during defecation. This is sufficient to prevent easy evacuation of feces, which are consequently retained.

It seems likely that the improvement following sympathectomy has been the result of the spinal anesthetic used for the operation or for diagnostic purposes. There is no excuse for performing sympathectomy. A spinal anesthetic should be used in place of general anesthesia when difficulty is experienced in emptying the rectum and pelvic colon by the use of enemas alone.

ANSPACH, Chicago.

TREATMENT OF INFANTILE DIARRHEA WITH PECTIN. FRANCISCO J. MENCHACA and NICOLAS ALBARRACIN, Arch. argent. de pediat. **21**:193 (March) 1944.

A brief summary of the literature regarding the mechanism of action of the diet of apples in infantile diarrhea and other intestinal conditions is presented. Seventeen infants having diarrhea were treated with 10 per cent pectin-agar-maltose-dextrin in skimmed milk. Excellent results were observed in those children who took the mixture well. Benefits were noticed even in some infants for whom this therapy had been used only a short time. Some infants refused to take the mixture at all.

CASSORLA, San Francisco.

Nervous Diseases

POLYNEURITIS OF UNKNOWN ETIOLOGY IN CHILD... ALEXANDER HATOFF, J. Pediat. **24**:393 (April)

The extensive literature on polyneuritis, polyradi... neuritis and the Guillain-Barré syndrome (virus ... cephalomyelitis) is reviewed. The histories of ... patients under 20 years of age whose symptoms ... form with those described for polyneuritis of unk... cause are presented. For further evaluation, 2 ... cases are presented, 1 in which polyneuritis followed ... administration of sulfathiazole and 1 in which it follo... diphtheria.

The consideration of this group of cases leads ... the conclusion that polyneuritis of unknown cause ... lows the same pattern in children as in adults. ... following differences are observed, however. ... patients outnumber female patients 3 to 1. ... average age of children with this malady is 9 y... The onset usually occurs during cool weather. A ... tory of a preceding illness is always obtainable, ... doubt is cast on the causal relationship between the ... Dysphonia and dysphagia are observed in addition ... paralytic phenomena. Tenderness in the muscle... usually not observed.

This disease is not nearly so benign as the literat... would lead one to believe. Polyneuritis of unkn... cause follows the same course as polyneuritis of kn... cause. The use of a proper name, such as Guill... Barré syndrome, for polyneuritis of unknown cau... not justified and will not be justified until this gr... of conditions can be differentiated from others on ... etiologic or pathologic basis.

HATOFF, Richmond, Cal...

INFLUENZAL MENINGITIS. STANLEY S. LAMM ... BERNARD H. SHULMAN, J. Pediat. **24**:408 (Ap... 1944.

This is a review of the cases of 15 patients ... influenzal meningitis treated in the pediatric ward ... the Kings County Hospital (Long Island College ... Medicine Division) and the Long Island College H... pital during the six years from 1938 to 1943.

Nine of the 15 patients recovered. Treatment ... of the patients consisted of the combined use of che... therapy (sulfapyridine), type-specific anti-influenzal ... bit serum and transfusions. Seven of the 8 recove... Two cases are reported in detail, 1 in which the patie... condition was complicated by agranulocytosis resul... from the chemotherapy (sulfadiazine and sulfapyrid... and 1 in which an apparent recovery was followed ... a relapse after chemotherapy was discontinued.

A prepared plan of therapy from the moment ... diagnosis is made, including the administration of d... and rabbit serum and blood transfusions, is outlined ... detail. The value of a prepared routine is evide... by the number of recoveries obtained in the age gr... under 2 years.

LAMM, Brookly...

ACUTE ATAXIA OF UNKNOWN ORIGIN IN CHILD... WALTER O. KLINGMAN and RICHARD G. Ho... J. Pediat. **24**:544 (May) 1944.

Seven cases of acute ataxia of unknown origin ... children are reported. Three patients in this group ... a history of acute febrile illness shortly before ... appearance of neurologic signs. Four of the patie... had been in good health up to the time of appear... of the ataxia. The predominant characteristics of ... illness were ataxia and tremor. Convulsions w...

ent at the onset in 1 patient. Recovery from the ologic symptoms was complete in 5 of the patients, there was very little residual impairment in coordi- n in the other 2. A marked conduct disturbance loped in 1 patient. We believe that the chief rtance of the recognition of this syndrome in this group lies in its differentiation from cerebellar lasm.

[AUTHORS' SUMMARY.]

TANEOUS SUBARACHNOID HEMORRHAGE IN IN- ANTS AND ITS RELATION TO HYDROCEPHALUS. . GORHAM BABSON, J. Pediat. **25**:68 (July) 1944.

hree cases of apparently spontaneous subarachnoid orrhage occurring between the ages of 18 and 30 of age are reported. Symptoms and signs of ingeal irritation with increased intracranial pressure n abruptly and with severity. The bleeding and r symptoms gradually abated with spinal drainage. of the patients later had symptomless hydrocepha- Subarachnoid bleeding, such as is described, is at this age. Bleeding of this nature due to injury irth or to occasional hemorrhagic disease of the born occurs during the first week of life. No vy, leukemia, hemophilia, purpura or infectious dis- was present. A rupture of a congenitally weak bral vessel may have been the cause in these cases. hydrocephalus presumably occurred as the result dhesions between the arachnoid and pial layers pro- d by the hemorrhagic reaction on the meningeal branes, with consequent obstruction to the free flow rebrospinal fluid. It is suggested that unrecognized rachnoid hemorrhage at birth is a potent cause of ing degrees of unexplained hydrocephalus. Free oval of blood should be instituted in such cases.

BABSON, Portland, Ore.

UMOCOCCIC MENINGITIS. ROBERT SCHWARTZ, New York State J. Med. **43**:1049 (June 1) 1943.

girl aged 4½ months had pneumococcic meningitis wing acute otitis media. She recovered after the of sulfapyridine and antipneumococcus serum (rab- type 33, supplied by the New York State Depart- t of Health.

AIKMAN, Rochester, N. Y.

NINGITIS DUE TO HAEMOPHILUS INFLUENZAE: RE- IEW OF TREATMENT. A. G. NICHOLSON, M. J. Australia **1**:320 (April 8) 1944.

icholson presents a brief review of the treatment of enzal meningitis. Since the introduction of sulfon- de therapy in 1939 the mortality rate at the Chil- 's Hospital in Melbourne has been 79.1 per cent. wever, only 4 patients have been treated by the tie Alexander method. Of these, 2 recovered and ed. Of the latter, one was only 8 weeks of age, in the other, aged 16 months, the disease was com- ated by gross bilateral pneumonia.

GONCE JR., Madison, Wis.

Psychology and Psychiatry

GULAR FORTY TO FIFTY DAY CYCLE OF PSYCHOTIC BEHAVIOR IN A FOURTEEN YEAR OLD BOY. KATHERINE K. RICE, Arch. Neurol. & Psychiat. **51**:478 (May) 1944.

After pointing out the relationship of cyclic behavior glandular disturbance produced by surgical interven- , the author describes a 14 year old boy who showed "nearly regular cycles" of psychotic behavior. He had a manic-depressive psychosis.

BEVERLY, Oak Park, Ill.

A STUDY OF THE GROWTH OF THE DYNAMIC CONCEPT OF KNOWLEDGE. DON C. FITZGERALD and RALPH H. OJEMANN, Child Development **15**:53 (March) 1944.

In this paper the results obtained in an analysis of the growth of the dynamic concept of knowledge in a group of 176 high school juniors and seniors are presented. A test for measuring various aspects of the concept as it applies to physical, physiologic and mental-social problems is described. The coefficient of reliability obtained by several methods varied from 0.72 to 0.79.

This group of high school juniors and seniors obtained a score in the neighborhood of 50 per cent of the total possible score. Since the test items were constructed by using everyday situations, it is apparent that the development of the concept had not proceeded far in this group.

When the applications of the concept to the three areas, physical, physiologic and mental-social, were compared, a significantly greater development in applications to the physiologic area was found. The physical and mental-social areas were about equal in terms of total possible score, but the development in the physiologic area was significantly higher. The cause for this difference is not at present apparent.

The amount of training in courses that might be expected to influence the development of this concept, namely, the number of semesters of science and of mathematics, was not significantly correlated with the scores. The coefficient of correlation between the number of semesters of science and the total score was 0.07 ± 0.08. Matched pairs of scores yielded no significant differences when compared in terms of number of semesters of mathematics taken.

Since the number of subjects achieving 75 per cent or more of a total possible score is extremely small, the next step in this series of investigations seems to be to develop experiences which will accelerate the development of the concept so as to make possible studies of the effect of the concept on the development of behavior and personality.

SPEECH CHARACTERISTICS OF PSYCHONEUROTIC PATIENTS. I. PETER GLAUBER, J. Speech Disorders **9**:18 (March) 1944.

The histories of 10 psychoneurotic patients are presented, together with a number of psychoanalytic observations on other patients. The author states that in "orally dependent" psychoneuroses there is a definite increase in the volume of speech, pauses are short and infrequent, speech is difficult to interrupt and the conversation includes much trivia and much circumstantiality. In "compulsive speech" the volume and rate are both increased, enunciation is much sharper than in the "orally dependent" type and emphasis is on the intellectual rather than the emotional plane. A speech disorder is a reflection of a disorder or idiosyncrasy of the total personality.

PALMER, Wichita, Kan.

STUDIES IN THE PSYCHOLOGY OF STUTTERING: XVIII. THE CONSTRUCTION AND APPLICATION OF A TEST OF ATTITUDE TOWARD STUTTERING. ROBERT AMMONGS and WENDELL JOHNSON, J. Speech Disorders **9**:39 (March) 1944.

A scale for the measurement of attitude toward stuttering, consisting of one hundred and sixty items

with five possible responses to each, was constructed and administered to 40 freshmen in college, 40 townspeople, 72 other persons who stuttered and 67 clinicians, 11 of whom were stutterers. The four groups differed a great deal from each other in their attitude toward stuttering. The clinicians had the least unfavorable reaction, freshmen and stutterers a moderate reaction and townspeople the most unfavorable reaction. No relation was found between the attitude toward stuttering and the severity of stuttering or the number of years of remedial speech work. Both sexes tolerated stuttering by men well. Men, however, were significantly less tolerant than women of stuttering by women. Items had different values when they were stated positively and negatively. Context in the form of adjacent items did not appear to have any effect on the mean value of items.

PALMER, Wichita, Kan.

AN ANALYSIS OF CHILDEN'S EATING HABITS. ALFRED L. BALDWIN, J. Pediat. **25**:74 (July) 1944.

On the basis of interviews with mothers 76 children were rated on three variables, appetite, finicalness and behavior at table. Supplemental information was available on the following points: (1) the character of the children's homes, including such traits as strictness of discipline, affectionateness of the parents and parental understanding of the children; (2) the personality traits of the children as exhibited in their adjustment in the nursery school. Good appetite and good behavior at table were generally found in children from homes in which strict disciplinary methods were combined with approval of the children.

The parents of children with good appetites seemed to be strict about a few essentials of behavior, but they did not completely regiment the children's activities. This type of strictness seems to be psychologically healthful, as is evidenced by the good adjustment these children made in nursery school.

The strictness found in the homes of children with good behavior at table seemed to stem from an attempt on the part of the parents to force the children into complete conformity with adult standards. This excessive restraint of children's behavior hampered their social adjustment.

Acceptance of a wide range of foods and lack of finicalness were most generally obtained by an abundance of affection and interest and not by coercive methods.

BALDWIN, Yellow Springs, Ohio.

A PERSONALITY STUDY OF STUTTERERS AND NON-STUTTERERS. LAVANGE HUNT RICHARDSON, J. Speech Disorders **9**:152 (June) 1944.

Thirty stutterers, 8 females and 22 males, who were members of a speech correction class at the University of Southern California, were studied by means of four tests of personality: the Henmon-Nelson tests of mental ability, form A, the Guilford inventory of factors S T D C R, the Rorschach ink blot test and the Murray thematic apperception test. The controlled group of 30 nonstutterers were matched with the stutterers for age (plus or minus five years), sex, decile rating on the Henmon-Nelson test of mental ability and college experience. The limitations of the various tests are discussed. A common statistical method is not feasible in interpreting the results, since the tests interpret different things. The author states that the Rorschach test is not amenable to statistical treatment or qualitative evaluation. On the inventory of factors S T D C R, the stutterers are more socially introverted, more depressed and less happy-go-lucky than the n stutterers. On the Rorschach test the main differe appears to be in the stutterers' failure to see moveme the author indicating that this is a failure to resp impulsively to outside environment. There are no di or movement responses in the Rorschach test. On thematic apperception test there are no significant d ferences among the groups. The author states that Rorschach and similar tests are valuable for furt study of individual stutterers.

PALMER, Wichita, Kan.

STUDIES IN THE PSYCHOLOGY OF STUTTERING: XI OLIVER N. BLOODSTEIN, J. Speech Disorders **9**: (June) 1944.

Thirty stutterers ranging in age from 17 to 42 ye 27 male and 3 female, were studied with the test oral reading rate which have been standardized Darley. Each subject read three 300 word passages factual prose. One of these passages was compo predominantly of polysyllabic words, one was compo entirely of monosyllabic words and one was carefu constructed to meet the requirements of "average" pr Two observers marked the reading time and stutteri of each subject. Only those spasms which were agr on by both observers were counted. The reliability the study was carefully studied by various statisti formulas. The stutterers read 122.7 words per min as compared with the mean reading rate of 167.3 wo per minute of nonstutterers determined by Darle earlier study. The apparent deviations of the distri tions of rate for all passages were found to be grea for stutterers than for nonstutterers. Stutterers, i nonstutterers, varied most widely in reading the mo syllabic passage and least widely in reading the po syllabic passage. The mean nonstuttering, fluent int val rate for the average passage was 144.7 words p minute. This is significantly faster than the mean ov all rate for stutterers. High negative correlations w obtained between over-all reading rate and both fr quency and total duration of stuttering. There is a a high negative correlation between these measures severity and nonstuttering rate. The author sugge that there may be a subtle psychologic difference experimental conditions for the two groups. Stutterer according to the author, may be retarded in oral rea ing rate by an anxious anticipation of stuttering. T author prefers this interpretation to one involving neur muscular instability. The author reviews the work others on this same problem and states as a final co clusion that the relation between the severity of stu tering and the rate of reading and probably of speakin is not a simple one. There are many complicati factors which have been presented both in this stu and in previous studies.

PALMER, Wichita, Kan.

Diseases of the Ductless Glands; Endocrinology

THE UPTAKE OF RADIOACTIVE IODINE BY THE THYROI OF RATS MADE GOITEROUS BY POTASSIUM THI CYANATE AND BY THIOURACIL. RULON W. RAWSO JOHN F. TANNHEIMER and WENDELL PEACOC Endocrinology **34**:245 (April) 1944.

When radioactive iodine was administered to ra four hours before they were killed the average uptak by the thyroids was as follows: In normal control 56 per cent of the given tracer dose; in rats made go

s by thiocyanate, 87 per cent, and in rats made ous by thiouracil, 10 per cent.

hen potassium thiocyanate is administered the ufacture of thyroid hormone is diminished, and rs resulting from treatment with this agent have a ter than normal avidity for iodine. Thiouracil acts lock the usual easy and ready uptake of iodine by thyroid. It appears that at least one action of this on the function of the thyroid is to interfere with iodinization of the thyroglobulin molecule, thus enting the elaboration of any physiologically active none.

JACOBSEN, Buffalo.

EFFECT OF SMALL DOSES OF DIETHYLSTILBESTROL N THE ANTERIOR HYPOPHYSIS OF THE IMMATURE RAT. BURTON L. BAKER and NEWTON B. EVERETT, Endocrinology **34**:254 (April) 1944.

is concluded that diethylstilbestrol exerts a stimue action on the acidophil cells of the anterior lobe he pituitary.

JACOBSEN, Buffalo.

EFFECT OF THIOURACIL ON THE FORMATION OF THYROXINE AND DIIODOTYROSINE BY THE THYROID GLAND OF THE RAT WITH RADIOACTIVE IODINE AS INDICATOR. A. J. FRANKLIN, S. R. LERNER and L. L. CHAIKOFF, Endocrinology **34**:265 (April) 1944.

is shown that the feeding of thiouracil to the rat rferes with the incorporation of injected radioactive ne into thyroxin and diiodotyrosine by its thyroid nd.

oth the capacity of the gland for concentrating ioactive iodine and the amounts of radiothyroxin radiodiiodotyrosine formed in the thyroid were reed to normal two weeks after the administration of uracil had been discontinued.

JACOBSEN, Buffalo.

FECT OF THIOURACIL ON THE IODINE CONTENT OF THE THYROID GLAND. E. B. ASTWOOD and ADELE BISSELL, Endocrinology **34**:282 (April) 1944.

The administration of thiouracil to young rats was lowed by a nearly complete disappearance of iodine m the thyroid glands in five days and a threefold rease in the size of the glands in two weeks. These ects were inhibited by hypophysectomy and also by injection of thyroxin.

The data support the view that thiouracil inhibits the rmation of thyroid hormone.

JACOBSEN, Buffalo.

Diseases of the Muscles, Bones, Joints and Lymph Glands

MYOPLASIA CONGENITA CAUSING MALPRESENTATION OF THE FETUS. MILDRED I. EALING, J. Obst. & Gynaec. Brit. Emp. **51**:144 (April) 1944.

Amyoplasia congenita, a rare condition, may cause mplications during labor because of spasticity of the al joints and associated hydramnios.

A report of a case with this complication is reviewed. The cause is unknown, and the disease is characrized by congenital symmetric rigidities of joints of rying degree, unassociated with inflammatory change. No specific pathologic change has been found in the ntral nervous system. Usually the mental condition normal.

CUMMINGS, Evanston, Ill.

ADOLESCENT KYPHOSIS. T. J. B. A. MACGOWAN, Lancet **1**:211 (Feb. 12) 1944.

The author believes that in every case of adolescent kyphosis the vertebral nucleus is destroyed before the ossification of the vertebral body is complete. This destruction may be caused by failure of the cartilaginous disk to retain the nucleus or by softening of the vertebral body which allows the nuclear tissue to prolapse into the soft vertebral body. If the patient is adolescent the epiphysis will be subject to trauma. Abnormal ossification follows.

From a review of 100 cases the author has come to the conclusion that patients with kyphosis can be divided into five types on the basis of the cause of the condition:

1. Thin, asthmatic, "debilitated" children. The external forces are normal, but there is decreased spinal resistance.

2. Fat, overgrown, "endocrine" or gigantic children. The external forces are normal, but the spinal resistance is decreased.

3. "Congenitally" abnormal children. The external forces are normal or increased while the spinal resistance is normal.

4. Tall, well built, precociously muscled, "apprentice type" children. The external forces are increased and the spinal resistance is normal.

5. Children with traumatic kyphosis. The abnormality follows an accident which has caused excessive strain on the spine. The external forces are increased, and the spinal resistance is normal.

LANGMANN, New York.

Diseases of the Eye

ANGIOMATOSIS RETINAE (VON HIPPEL'S DISEASE): RESULTS FOLLOWING IRRADIATION OF THREE EYES. F. C. CORDES and O. C. DICKSON, Am. J. Ophth. **26**:454 (May) 1943.

Cordes and Dickson supply the following summary:

"Three eyes of two patients with angiomatosis retinae were irradiated. In the first patient, who had an early lesion in one eye, there was a marked improvement with retention of 0.8 vision, 3½ years after irradiation. In the second case both eyes were involved, the right eye being in a well advanced stage, whereas the lesion in the left eye was an early one. Both eyes were given 1,800 r. The early lesion showed definite improvement in vision of 1.0 two years after irradiation. During this time the eye having the more advanced lesion became progressively worse, going on to amaurosis, complete detachment of the retina and gliosis.

"From our experience with three eyes and from what is reported in the literature it appears that in early cases X-ray therapy does offer a convenient, safe means of treating these lesions. The advanced lesions have not responded to any type of therapy.

"Electrolysis, diathermy, radium, and x-ray irradiation have all been used with success. It seems worthwhile to record the results obtained so that at a later date it may be possible to evaluate better the various methods now employed in the treatment of this rare disease."

The article is illustrated.

W. ZENTMAYER. [ARCH. OPHTH.]

JUVENILE CATARACT IN ASSOCIATION WITH DERMATOSIS. D. M. ROLETT, Am. J. Ophth. **27**:389 (April) 1944.

Rolett reaches the following conclusions with regard to the disease described by Rothmund:

"The important points to be remembered are:

"1. Occurrence of cataractous changes in rather young people with typical skin changes which may or may not arise simultaneously.

"2. Evidence of pluriglandular endocrine disturbances.

"3. Skin changes although bearing a similarity are not always of the same origin and are not therefore the cause of the disease.

"4. The development of the cataract is rapid and progressive.

"5. The prognosis as to the operation and subsequent vision is favorable.

"6. Fortunately the disease is not common."

W. S. REESE. [ARCH. OPHTH.]

CASE OF NEUROFIBROMA OF THE CHOROID. P. D. TREVOR-ROPER, Brit. J. Ophth. **28**:177 (April) 1944.

A girl aged 3½ years had moderate proptosis of the left eye, with limitation of all movements. Ophthalmoscopic examination showed a globular mass almost filling the superotemporal quadrant; this was whiter than the adjacent choroid, with distinct choroidal blood vessels on its surface and normal retinal blood vessels directly overlying it. There was no proliferation of pigment. The mass was sharply demarcated and almost reached the optic disk; at the lower border was a trace of fluid detachment. Exploration of the orbit exposed a firm, circumscribed tumor extending back from the sclera toward the apex of the orbit. The orbit was exenterated.

The tumor had a well defined fibrous capsule, although this was poorly developed over the intraocular portion. The growth was composed of elongated cells, showing well marked parallel arrangement and many areas of palisading. It was unusually cellular, and there were a few mitotic figures. Collagen formation was correspondingly scanty, but well developed fibrous septums could be seen, and there was some collagen in the cell bundles. Small areas of hemorrhage and degeneration were present. In spite of the cellularity, the general appearance did not suggest a malignant growth. The tumor belonged to the group of perineural fibromas. Its situation in the orbit was unusual, and it appeared to have arisen from one of the ciliary nerves, probably from the intrascleral portion; this would explain the intraocular and extraocular extensions of a noninvasive tumor.

The author could find but 6 recorded cases of discrete neurofibroma of the choroid; as in the case reported, the majority of the tumors were in a position suggesting their origin from the ciliary nerve, although in 1 case such an association was sought in vain. In common with the other cases, there was in the author's case no evidence of generalized Recklinghausen's disease, no family history and, as yet, no recurrence.

Diffuse neurofibromatosis of the choroid is more frequent in infants and usually results in glaucoma; it is generally associated with evidence of Recklinghausen's disease elsewhere.

The article is illustrated.

W. ZENTMAYER. [ARCH. OPHTH.]

Skin Diseases; Allergy

DERMATOPATHIC LYMPHADENITIS IN INFANTILE [E]ZEMA. VINCENT DE PAUL LARKIN, PAUL DISANT AGNESE and MAURICE N. RICHTER, Pediat. **24**:442 (April) 1944.

Lymphadenopathy has been frequently noted [in] patients with eczema; the condition may be local[ized] or generalized. It is dependent on the presence [of a] chronic cutaneous eruption; frequently the cutane[ous] disorder is characterized by pruritus, and it is [often] associated with eosinophilia. Six patients with infan[-]tile eczema and lymphadenopathy are described by [the] authors. A biopsy of inguinal or cervical lymph [node] was performed for each patient. Microscopic examina[-]tion revealed in all of them preservation of the ba[sic] architecture of the lymph nodes, together with the fol[-]lowing characteristic changes: replacement of all [or] part of the lymphatic tissue with proliferated reticul[um] cells, histiocytes and fibroblasts and deposition of [iron] and melanin. Clinically some of these patients may [be] suspected of having a blood dyscrasia, but the condition is benign.

LARKIN, New York.

Surgery and Orthopedics

THE PREVENTION OF HOSPITAL INFECTION OF WOUN[DS]. C. WHITE, M. J. Australia **1**:126 (Feb. 12) 1944.

The sources of infection in a hospital are so numer[-]ous that many people, from surgeon down to junior nurse, are concerned in protecting the patient from cross infection. A responsibility which is shared so widely tends to be shelved by all. Some English hospitals have therefore appointed one person to the job of teach[-]ing and maintaining a proper technic of asepsis throughout the hospital. In the early stages, at any rate, the job is a full time one.

GONCE, Madison, Wis.

COMPLETE ANTERIOR DISLOCATION OF THE ELBOW WITHOUT FRACTURE. T. MCHUGH, South African M. J. **18**:84 (March 11) 1944.

The author reports an anterior dislocation of the elbow joint without fracture in an 11 year old boy. The dislocation, which was produced by a fall from a donkey, could be corrected only by open reduction (posterior approach).

GONCE JR., Madison, Wis.

Teeth and Dentistry

THE TEETH OF SCHOOL CHILDREN FROM THE POINT OF VIEW OF THE SCHOOL DOCTOR. CHRISTOPHER ROLLESTON, Brit. J. Child. Dis. **40**:31 (April-June) 1943.

This study is based on the observation of 2,096 children examined by the same observer throughout their school life from 4 to 14 years. The number of children with excess caries, i. e., with 4 or more decayed teeth at any period of their school life, was 1,026 (48.9 per cent). The sexes were equally affected.

The author concludes that there is no connection between dental caries and any of the following conditions: malnutrition, enlarged tonsils, adenoids and thyroid gland, digestive troubles and rheumatism.

There seems to be no difference as far as the incidence of dental caries is concerned between breast-fed and bottle-fed children.

The employment of dental hygienists is strongly recommended for children of and under school age.

LANGMANN, New York.

Miscellaneous

MPARATIVE TOXICITIES OF SULFADIAZINE AND SULFATHIAZOLE IN CHILDREN. JAMES O. DOWRIE and MASON H. ABRAMSON, J. Pediat. **24**:176 (Feb.) 1944.

The toxic reactions observed in a group of 54 children ated with sulfadiazine were compared with those obrved in a group of 53 children treated with sulfathiae during approximately the same period. Granulotopenia occurred in 44.3 per cent of the children in first group and in 37.8 per cent of those in the cond. There was no agranulocytosis, even when adnistration of the drug was continued after granulotopenia appeared.

Simultaneous administration of alkali was employed the majority of instances. Gross hematuria occurred only 1 child, who had not received alkali. No inances of anuria were observed. The use of alkali pears greatly to lessen the danger of complications the urinary system.

Drug rashes occurred four times, always in children ceiving sulfathiazole. In 3 children the rashes rembled erythema nodosum.

The administration of ascorbic acid in massive doses peared to have no detoxifying effect for the two lfonamide drugs studied.

Observation of the effects on children failed to reveal y striking difference in toxicity between sulfadiazine d sulfathiazole with the exception of the greater inidence of rashes with the latter.

FROM THE AUTHORS' SUMMARY.

LINICAL TESTS OF CELLULOSE ACETATE PHTHALATE AS AN ENTERIC COATING. HAROLD C. HODGE, HENRY H. FORSYTH JR. and GEORGE H. RAMSEY, J. Pharmacol. & Exper. Therap. **80**:241 (March) 1944.

The authors note that cellulose acetate phthalate has e four qualities necessary for a substance that is to act a covering for enteric capsules: 1. It is extremely soluble in acid solutions. 2. It is rapidly soluble in lute alkaline solutions. 3. It will form a coating film. It is nontoxic. In a series of roentgen studies on edical students given barium sulfate in such capsules was found that 80 to 100 per cent of the capsules ere disintegrated in the gastrointestinal tract in the ght hours after ingestion (after breakfast or lunch). he average was four hours; only a few remained intact ore than eight hours. Only 1 capsule disintegrated in e stomach. In the succeeding article in the same numer of the journal (page 250) it is shown that the llulose acetate phthalate was nontoxic for rats and gs when given by mouth.

PILCHER, Cleveland.

Miscellaneous

HARMACOLOGICAL ACTION OF THE VENOM OF LATRODECTUS MACTANS AND OTHER LATRODECTUS SPIDERS. RAFAEL R. L. SAMPAYO, J. Pharmacol. & Therap. **80**:309 (April) 1944.

This excellent article from Buenos Aires, Argentina, tates that poisoning from the bite of this genus of ider is not uncommon in the United States as well s in other countries. Clinically the symptoms are due o the action of the toxin on the central nervous system, ncluding the autonomic system.

"The bite of Latrodectus mactans causes in man a ypical syndrome from which the diagnosis can readily be made, even without knowledge of the bite. With the bite a small puncture can be seen. After a latent period of about a quarter of an hour a burning pain is felt in the bitten area, and this soon spreads over the whole body. The pain becomes of an agonizing character and is accompanied by a state of mental irritability and restlessness. In addition, tremors and clonic contractions are noted, appearing first in the bitten area and later extending throughout the whole body. Pain and neuromuscular manifestations are intermittent and slowly grow in intensity. As the poisoning progresses, there come spasmodic movements of the legs, abdominal rigidity, marked salivation and lacrimal secretion, exhausting sweating, the sensation of precordial oppression and fear of imminent death. There are noted hyperesthesia, exaggerated reflexes, usually tachycardia and hypertension. Among other effects very frequently observed are intestinal and vesical palsies, albuminuria and oliguresis. Occasionally priapism, seminal emissions and enuresis occur. All of these symptoms lessen in a few hours but return to some degree on the following days, lasting altogether for from 48 to 72 hours. Throughout the poisoning, the local effects are insignificant—a small red puncture spot and sometimes a localized edema."

An antitoxic serum was obtained by immunizing horses with the toxin from the glands of the spider. Relief from the poisoning was established within three hours. Extensive pharmacologic studies, including electroencephalograms, were made in the investigation. Twelve thousand spiders were used.

PILCHER, Cleveland.

ROENTGEN STUDY OF THE FETUS IN UTERO: SOME PRACTICAL CONSIDERATIONS. WILLIAM SNOW and MILTON NADEL, Radiology **42**:136 (Feb.) 1944.

When sufficient lime is deposited in the bones to permit visualization in utero, the fetus is from 12 to 14 weeks old. Later the size of the image is used to estimate the age. Results obtained by using the occipitofrontal diameter parallel those obtained by using the perimeter of the head in computing size and age. The weight can be estimated by correlation of (1) the size of the fetus as shown on the film, (2) the size of the bones, (3) the perimeter or diameter of the skull, (4) the amount of subcutaneous fat (best seen over the breech) and (5) the appearance of the centers of ossification. Because of the inaccuracy of the determinations the authors prefer to omit actual numbers and use the terms small (5½ to 6½ pounds [2,500 to 2,950 Gm.])., medium (6½ to 7½ pounds [2,950 to 3,400 Gm.]), large (8 pounds [3,630 Gm.]) and very large (9 pounds [4,080 Gm.]).

Fetal death may be revealed by roentgen studies. Overlapping of sutures of the skull before labor is the earliest sign; later abnormal curvature of the spine, shrinking or even disintegration may be evident. The formation of a lithopedion is rare.

Abnormal positions of the fetus seen in roentgenograms may be due to placenta previa, fibroids, abdominal pregnancy or hydramnios.

Extension or deflection of the fetal head may furnish important information. Hyperextension before labor may be caused by the umbilical cord around the neck, hydramnios, placenta previa, pelvic masses, distention of the bladder or rectum, presence of twins, a tumor in the neck of the fetus, occiput anterior position of the fetus, pendulous abdomen and abdominal pregnancy. The most common causes of hyperextension or flexion

of the head during labor are cephalopelvic disproportion or occiput posterior position of the fetus.

Engagement of the fetal head may be prevented by one of the aforementioned conditions, usually by cephalopelvic disproportion.

Monstrosities are usually readily recognizable in roentgenograms. Acrania and anencephaly, usually associated with hydramnios, are the most common abnormalities. Elephantiasis congenita can be recognized by the extensive deposits of fat about the fetus. In a symmelus, or "mermaid," there is fusion, of various degrees, of the bones of the lower extremities. Extensive deformities with fractures are present in a fetus with osteogenesis imperfecta and great enlargement of the head in a fetus with hydrocephalus. Spina bifida, hemivertebrae, supernumerary digits, syphilis, rickets and fractures may be recognized in utero by roentgen studies.

ANSPACH, Chicago.

ON GROWING UP. MATTHEW WHITE, Glasgow M. J. **23**:37 (Feb.) 1944.

The author points out that the child is an actively growing organism and that this fact has far reaching influences on physiologic and pathologic processes. The reaction of the growing child to injury is immediate and effective, with a strong trend toward restoration of normal structure. This trend is most evident in the repair of fractures. Resistance to infection is acquired slowly by children. The introduction of the sulfonamide drugs has improved the outlook with regard to infections in children. Once the infection has been combated, functional recovery is much better in children than in adults.

There are three periods of accelerated growth: (*a*) infancy, culminating with the acquisition of the first dentition; (*b*) the interval from the end of the fifth year to the time of acquisition of the second dentition, and (*c*) puberty. Each of these periods of accelerated growth is accompanied by anatomic, physiologic and psychologic changes and is a period when instability and disturbance of structure, function and health apt to occur. The author speaks of comparative velopment in other mammals and shows that one not necessarily draw analogies from them; for exa the rat at birth is comparable to a 4 month hu fetus and a guinea pig at birth to a 7 year old c The accelerated growth at puberty often produces structural deviations as rounded shoulders, forward and flat feet. There are rapid skeletal growth and muscular tone. Wryneck often appears at birth to a hematoma of the sternocleidomastoid muscle, may appear at the age of 5 years, when rapid ske growth brings out the effect of a congenitally short muscle. Some systems mature faster than others. lymphatic system is much more pronounced in c hood; hence the greater incidence of adenitis, par larly mesenteric adenitis, during that period. The e crine glands also have different periods of effect growth. The growth of the limbs is proportiona greater than that of the trunk in childhood. Infection the bone often bring about stimulation of growth, whe injury to the epiphysial cartilage will cause delay growth. The rate and completeness of structural re are greater, the younger the child. The capacity restoration of function is much greater in the yo child.

SNELLING, Toronto, Canad

PEDIATRIC NEUROLOGY IN ART. F. ESCARDO, A argent. de pediat. **21**:257 (March) 1944.

The author analyzes from the pediatric and neurol point of view several masterpieces of various fam painters. He cites, for example, the hemiplegia Ribera, the myxedema of Velasquez and the dwarf of Zuloaga.

He mentions patterns of a pseudo Babinski s microcephalia, the Magnus reflex and other anoma as painted by several artists. He feels that car examination of these masterpieces is a valuable sou of knowledge.

CASSORLA, San Francisc

Book Reviews

ocrinology in Woman. By E. C. Hamblen, B.S., M.D., F.A.C.S. Price, $8.00. Pp. 571, with 157 llustrations. Springfield, Ill.: Charles C Thomas, 1945.

hen development in a field of medicine proceeds as dly as endocrinology has done in the past dozen rs it is inevitable that enthusiasm on the part of ratory workers, manufacturers and physicians should to conclusions not fully justified and often inaccu-. Perhaps it is a good thing, as the author suggests, as war has greatly limited productive work, one stop to assess the true value of what has been e.

he author has had considerable experience in endo-ologic investigation and in teaching. The book is ded into five parts, dealing with embryology and tomy, endocrine physiology, diagnostic methods, tional disorders and endocrinology applied to gyne-gic disease. Chapters will be found dealing with all glands.

he discussion of the anatomy and physiology of the ous glands is satisfying and instructive.

ibliographic references accompany each chapter. cussion of the various periods of the reproductive le form a part of the work. Methods of clinical mination are dealt with, and a satisfactory descrip-n of gynecologic studies of children is given. The pters devoted to treatment are especially pleasing. ere has been too much irresponsible and poorly docu-nted discussion of endocrine treatment for gynecologic orders. This has led to treatment of many patients o would have been better off had nothing in the form endocrine therapy been attempted. Many physicians too well informed themselves, have been influenced the claims of representatives of manufacturers. The blic, knowing nothing about it, has none the less n intrigued by the idea of "glandular treatment" by ans of injections. This has led to a far too free e of methods which intelligently employed are of eat benefit. Young girls with some unimportant viation from the average normal of the menstrual cle are brought to the physician for treatment by ans of "shots." Proper treatment of many of these tients consists in convincing the mother that the child eds nothing more than proper diet, rest and exercise. his sane view of gynecologic endocrinology, which is pported by the author, will appeal at once to all ex-rienced and conscientious gynecologists. Particularly, s denunciation of androgen is timely. While there ay be some difference of opinion as to whether this rmone should ever be used in treating females, many ysicians believing that it may occasionally be useful, ere can be no doubt that it has been far too widely ed.

A particularly pleasing chapter is the one on dysmen-rrhea. A dysmenorrheic girl is often like the woman the Gospel according to St. Mark who had "suffered any things of many physicians." The evaluation of e things which may be done, including surgical inter-ntion, is quite worth while. The great majority of xperienced gynecologists will agree with the rejection presacral sympathectomy for the relief of menstrual ain.

Chapters are included on disorders of the breast which may be regarded as due to endocrine imbalance; cutaneous abnormalities, and headache at the time of menstruation and other cyclic syndromes. Complications of pregnancy in which endocrine therapy may be useful and a useful chapter on sterility occupy the last portion of the book. In the concluding discussion of the climacterium the statement that bleeding which returns after the menopause should always be regarded as due to a malignant condition until proved otherwise will meet the approval of all those who have had considerable operative experience in gynecology. Too many women have been treated under precisely these conditions by the injection of glandular extracts by inexperienced or unwary physicians.

The book as a whole is one to which warm approval may be given. It will be most useful to the specialist, but the physician who meets, as all general practitioners do, some patients with functional troubles can turn to it for help. The printing and binding are good. The number of illustrations is moderate, but they are well done and give an excellent picture of the conditions they are intended to portray. The composition is excellent. The author expresses himself clearly and without needless words.

The Diseases of the Endocrine Glands. By Herman Zondek, M.D. Translated by Carl Prausnitz Giles, M.D. Fourth (second English) edition. Price, $11.00. Pp. 496, with 180 illustrations. Baltimore: The Williams and Wilkins Company, 1944.

This is the second English edition of a book already well established when its last German edition appeared in 1926. It is, according to the author's statement, primarily for the clinician. It is divided into two main sections. The first of these, comprising 118 pages devoted to a general survey of the functions of the endocrine system, the chemistry of hormones and methods of examination of patients, gives an extraordinarily condensed, complete and up-to-date picture of the subject as a whole. The second section deals with diseases, which are arranged without any effort to group them according to their glandular origin. This is done in order to avoid the necessity of placing a disease in a category associated with a specific gland, since it is the author's belief that the majority of the maladies of internal secretion are pluriglandular syndromes.

The chapters on sex glands and sex hormones are especially good. The discussion of exophthalmic goiter is exceptionally complete, requiring 60 pages of characteristically condensed writing. Indeed, throughout this book one has the impression that a two volume work has been boiled down into one without omitting any of the facts. It is evident that the author is writing from personal experience with the various conditions he discusses, and on this basis he seems willing to make definite commitments on matters which others might approach more cautiously. One cannot help getting the impression that he quotes Zondek rather too often.

From the point of view of the pediatrician there are some omissions and some subjects which are considered too briefly. No reference is made to the response of serum cholesterol to withdrawal of thyroid medication

in a hypothyroid child, nor to epiphysial dysgenesis. Simple goiter and endemic cretinism are considered as essentially one disease: "The morbid phenomena in both arise from the same degenerative constitution." Perhaps this is true of the type seen in Europe. In the chapter on tetany rickets is not even mentioned! Sudden death in a person with a large thymus is stated to be due to a toxic factor which is particularly active in persons of congenitally deficient constitution. The death of a nurse immediately after an intravenous injection of antitoxin is ascribed to severe anaphylactic shock because of her status thymicolymphaticus. There is an attempt to subdivide obesity into so many different types that the reader is left confused.

Dr. Zondek's book is of great interest and shows evidence of advanced thought and even foresight in the field of endocrinology. It is stimulating to the cover-to-cover reader and a valuable reference book to have handy on the desk.

Applied Dietetics. The Planning and Teaching of Normal and Therapeutic Diets. Second edition. By Frances Stern. Price, $4.00. Pp. 265. Baltimore: The Williams and Wilkins Company, 1943.

Dietitians, physicians, nurses, teachers, students, public health workers and social workers will find this book of inestimable value because it is factual, concise, thorough and sound. The practical procedure for planning normal and therapeutic diets and for teaching their preparation is based on methods evolved by the author during twenty-five years of experience with patients in the food clinic of The Boston Dispensary.

Those familiar with the first edition of this successful book will see that a wealth of material has been added in this revision. Results of recent nutrition research have been included and new data on vitamins incorporated in the text and tables.

The book logically considers the therapeutic diet merely a variation of the normal diet necessitated by certain pathologic conditions. Each step in the construction of both the normal and the therapeutic diet is developed with reference to the needs of the body; criteria for selection of food constituents in right amounts and for determining the amounts and kinds of foods needed to supply them are provided.

Environmental factors that influence the effectiveness of the diet and methods of educating the patient with the normal and the therapeutic diet are discussed. New illustrative material which will be helpful in teaching the patient is presented. In addition to the fifty-seven coordinated tables given to simplfy the computation of diets, there is a series of complete and concise outlines for the dietary treatment of various abnormal conditions. Of special interest are those covering the vitamin deficiencies. Typical diets are computed to show how the food prescription may be filled and suggested meal plans and menus of different costs are given for each diet.

"Applied Dietetics" will be of great value in organizing teaching material for the medical student, the student dietitian or the nurse as well as for the layman. Dietitians should find it invaluable.

Artificial Pneumothorax in Pulmonary Tuberculosis. By T. N. Rafferty, M.D. Price, $4.00. Pp. 192, with 26 illustrations. New York: Grune & Stratton, 1944.

The author of this valuable and interesting book on artificial pneumothorax was formerly resident physician at the William H. Maybury Sanitorium (the Detroit Municipal Tuberculosis Sanitorium), Northville, M

The book is divided into three main sections: ge considerations, choice of cases and management.

The various forms of collapse therapy are consid the greatest advance in the control of tuberculosis s the value of rest in bed was first appreciated. author points out however, that the available stati have often been misinterpreted, and that the valu collapse therapy will be more and more evident as t comes to be a more proper selection between c suitable for treatment by artificial pneumothorax those in which thoracoplasty or some other metho the treatment of choice. The advance in the techni closed intrapleural pneumolysis, without which an a quate collapse of the diseased portion of the lung often impossible, has added immeasurably to the va of treatment by artificial pneumothorax.

The careful details as to selection of cases and m agement, as well as an unusually keen analysis of literature, makes this study an outstanding contributi

The Art of Anesthesia. Seventh edition. By Pa J. Flagg. Price, $6.00. Pp. 519, with 166 illustr tions. Philadelphia: J. B. Lippincott Company, 19

This work, now in its seventh edition, is one wh has long been recognized as a standard textbook anesthesia. It is principally a plea for a better und standing of the art of the administration of ethyl eth As such, it is of more value to the general practition who is occasionally called on to give an anesthe than it is to the specialist in anesthesia. Much oth material is passed over lightly and given too li space. Other methods and agents are mentioned o to prove that ether or combinations of ether and g are more desirable and safer. The material on the u of ether is comprehensive and informative, but in t discussions of these agents there are some import omissions; for example, in the chapter on local an thesia there is no mention of toxic reaction to the dru precautions to be observed to prevent these reactions treatment if they occur.

The chapters on resuscitation and pneumatology co tain much valuable material. The chapters on pr operative and postoperative care, on evaluation of t patient's condition and on the choice of method a agent in various types of surgical procedures are exce lent. The book is an excellent review of the author experiences over a long period as a specialist in the a of anesthesia.

Character Formation Through Books: A Bibl ography. By Clara J. Kircher. Introduction Dom Thomas Verner Moore, O.S.B., Ph.D., M.D Price, not given. Pp. 79. Washington, D. C The Catholic University of America, 1944.

With the premise that a child lives out the role the hero of a book and thus obtains emotional relea as well as gleaning general principles of conduct fro the actions of the characters, the author has selected group of books for children from the primary ag group through adolescence. He gives a brief descrip tion of each book and enumerates the character trai and other topics emphasized in it.

The books selected for the primary group stress co tentment, good manners, generosity, helpfulness an repentance; some are on the subject of babies. Th books for the lower grades emphasize bravery, honest and manliness. Those for the middle grades teac simplicity, obedience and thoughtfulness; some deal wit

opics of sex education, fathers and prayer. Those nior high school suggest unselfishness, promptness, and honesty. The senior high school books cover subjects and others, such as running away from as against staying and going to the local teachers' ge in order to help one's father in the store another before going away to the university. Of the list whole one may say that a group of books has been ed whose purpose is to coerce children to accept standards: It is another form of stern discipline.

s on Nursing by a Nurse. By Sarah Corry, .N., graduate of Evanston Hospital School of ursing, affiliated with Northwestern University, vanston, Ill. Price, $1.50. Pp. 144, with 3 illustrations. New York: D. Appleton Century Company, 1944.

his book is dedicated to all nurses who know but have forgotten. It is based on the training given vanston Hospital School of Nursing. Pocket size convenient carrying, it is remarkably thorough, in of its condensation. Miss Corry suggests that it be used as a teaching guide in small hospitals, as rk of reference for persons who have changed their s in nursing and need to review a forgotten routine, s an aid in the event of an emergency in some ted area.

my opinion this handy book will be most valuable those who have not been nursing for some years and now, because of wartime necessity, are coming into this field. Much "brushing up" on old rous will be necessary, as well as catching up on new hods and medicines. The cost is a small outlay a great deal of valuable information, including disions of penicillin, treatment of poliomyelitis, emercies of all kinds, deliveries at home, antidotes to ons and war gases—to name a few of the topics ted.

ections of the Peritoneum. By Bernhard Steinberg, M.D. Price, $8.00. Pp. 455, with 45 illustrations. New York: Paul B. Hoeber, Inc., 1944.

This work shows the results of years of experimental practical study.

t not only takes up the normal structure and funcns of the peritoneum but combines an extensive study ough laboratory experimentation of the various ses of peritonitis with a study of its clinical behavior treatment.

The result in peritonitis—and this statement applies both infants and adults, inasmuch as the mechanisms olved are basically the same—depends on the timely cognition and treatment of the disease in its various ges.

The author determines the current stage by frequent dominal puncture to determine the number and kind bacteria relative to the number of phagocytic, polyrphonuclear leukocytes.

I believe this is Dr. Steinberg's main contribution to literature on peritonitis, although he does take up equately diagnosis and treatment by means of roentn rays, along with correction of chemical changes in body and use of chemotherapy, vaccines, antitoxins d certain mechanical measures.

This treatise on infections of the peritoneum opens w channels in the diagnosis and treatment of intradominal inflammatory lesions, both chemical and bacricidal.

An Outline of Tropical Medicine. By Otto Saphir, M.D., Director of the Department of Pathology of Michael Reese Hospital; Professor of Pathology, University of Illinois College of Medicine. Price, $1.00. Pp. 85, with no illustrations. Chicago: Michael Reese Research Foundation, 1944.

This small book is an outline covering the essential characteristics of the most common tropical diseases.

The table of contents includes the following subjects: virus diseases, rickettsial diseases, leishmaniasis and trypanosomiasis, treponemas and leptospiras, bacterial diseases, malaria and helminthic infections.

The book is intended to serve as a ready source of condensed information on tropical diseases. This purpose has been admirably achieved.

No illustrations accompany the text. A bibliography at the end of each section refers the reader to the larger textbooks on tropical diseases.

The author has succeeded in supplying essential information in a clear and concise manner.

Simplified Diabetic Management. Fourth edition. By J. T. Beardwood Jr. and J. D. and H. T. Kelly, M.D. Price, $1.50. Pp. 172, about half of which are devoted to tables and charts. Philadelphia: J. B. Lippincott Company, 1944.

According to the authors' preface, this book is designed primarily as a guide and source of reference for the diabetic patient. Also it offers the physician valuable data and plans for the formulation of diets for the adult diabetic patient. For the adult patient and for his physician the book will serve a useful purpose. The consideration of the diabetic child, however, is too brief for the inclusion of this topic, by inference, in the scope of the book.

Motivos de pediatría. By Alfonso G. Alarcón. Price not given. Pp. 332, with 15 illustrations. Mexico, Mexico: Imprenta Gallarda, I. A. Franco, 1944.

The various nutritional disturbances and the metabolic diseases of infancy are discussed from a physiologic and psychologic standpoint. Final chapters on the principles of the care of children in Mexico are of especial interest. This book, written in Spanish, should be of interest to one wishing to learn how such problems are handled in Mexico. There is a well chosen bibliography for most of the chapters.

Sobre distrofias en el lactante. By Calixto Torres Umana. Price not given. Pp. 287, with 8 colored plates. Bogota, Colombia: Libreria Siglo XX, 1944.

This is a textbook written in Spanish on digestive and metabolic disturbances in children. The various diseases are considered in detail, and the treatment used in Colombia is described. It is an interesting book. Rickets is rare in the high altitude of Colombia, but a photograph is shown of a patient with severe rickets encountered by the author. An excellent bibliography is appended.

The Daily Log for Physicians for 1945. Price, $6.00. Pp. 500, with no illustrations. Champaign, Ill.: Colwell Publishing Company, Not Inc., 1945.

"The Daily Log for Physicians" is again published. It contains the usual spaces for information and will help the average doctor to improve his bookkeeping for his income tax as well as serve many other purposes.

This log has become almost a necessity to many physicians.

Directory of Pediatric Societies *

INTERNATIONAL

International Association of Preventive Pediatrics

President: Prof. S. Monrad, Dronning Louises Børnehospital, Copenhagen, Denmark.
Secretary: Dr. Daniel Oltramare, 15 Rue Lévrier, Geneva, Switzerland.

International Congress of Pediatrics

President: Dr. Henry F. Helmholz, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Charles F. McKhann, University Hospital, Ann Arbor, Mich.
Canadian Committee:
Chairman: Dr. Alan Brown, Hospital for Sick Children, 67 College St., Toronto.
Secretary: Dr. H. P. Wright, 1509 Sherbrooke St. W., Montreal.
Place: Boston. Time: Postponed indefinitely.

International Congress for the Protection of Infancy

Secretary: Prof. G. B. Allaria, Corso Bramante 29, Torino 120, Italy.

FOREIGN

Argentine Pediatric Society of Buenos Aires

President: Dr. Martin Ramón Arana, 1809 Rodriguez Peña, Buenos Aires.
General Secretary: Dr. Alfredo Larguia, Cerrito 1179, Buenos Aires.

Associação Paulista de Medicina, Section on Pediatrics

President: Dr. Vicente Laro.
First Secretary: Dr. Armando de Arruda Sampalo.
Second Secretary: Dr. Luiz Augusto de Toledo, Av. Brigadeiro Luiz Antonio 393, 1° Andar, São Paulo, Brazil.
Time: Twelfth of every month, 8:30 p. m.

British Paediatric Association

President: Prof. L. G. Parsons, 58 Calthorpe Rd., Five Ways, Birmingham.
Secretary: Dr. Donald Paterson, 27 Devonshire Pl., London, W. 1.

Danish Pediatric Society

President: Dr. E. Lenstrup, Copenhagen.
Secretary: Dr. E. Gjørup, Dronning Louises Børnehospital, Copenhagen.

Nederlandische Vereeniging voor Kindergeneeskunde

President: Dr. J. H. G. Carstens, Servaasbolwerk 14ª, Utrecht.
Secretary: Dr. R. P. van de Kasteele, Laan van Poot 340, 's Gravenhage.
Place: Different places. Time: Three times a year.

Paediatrický Spolok na Slovensku

President: Dr. A. J. C. Chura, Lazaretská 11, Bratisl
Secretary: Dr. P. Rados, Lazaretská 6, Bratislava.
Place: Pediatric Clinic, University Bratislava. Ti
Six times year.

Royal Society of Medicine, Section for the Study of Disease in Children

President: Dr. E. A. Cockayne, 98 Harley St., Lond
W. 1, England.
Secretary: Dr. R. Lightwood, 86 Brook St., Lond
W. 1, England.
Place: 1 Wimpole St., London. Time: Fourth Fri
of each month, 4:15 p. m.

Palestine Jewish Medical Association, Section of Physicians of Children's Diseases

President: Prof. S. Rosenbaum, 26 Bialkstr., Tel A
Secretary: Dr. A. Brünn, 9 Maazestre, Tel Aviv.

Sociedad Cubana de Pediatria

President: Dr. Angel A. Aballí Arellano, 17 No.
Vedado, Habana.
Secretary: Dr. Julio G. Cabrera Calderin, Hosp
Mercedes L y 21 (Vedado), Box 2430, Habana.
Place: Cátedra de Clinica Infantil, Hospital Merce
Habana. Time: Last Wednesday of every month.

Sociedad Mexicana de Pediatria

President: Dr. Fernando López Clares, 12/a. Mede
191, Mexico.
Secretary: Dr. Jesus Gómez Pagola, Versalles
Mexico.

Sociedad Venezolana de Puericultura y Pediatr

President: Dr. E. Santos Mendoza.
Secretary: Dr. P. Oropeza, Hospital de Niños, Carac

Société de Pédiatrie de Paris

President: Dr. B. Weill-Hallé, 49 Avenue Raymo
Poincaire, Paris, France.
Secretary: Dr. Jean Hallé, 10 bis Rue Pré aux Cler
Paris, France.
Place: Hôpital des Enfants Maladies, 49 Rue de Sèvr
Time: 4:30 p. m., third Thursday of every mon

Uruguyan Society of Pediatrics

President: Dr. Jose Alberto Praderi, Eduardo Acev
1132, Montevideo.
Secretary: Dr. Hector Fossati, Juan D. Jackson 1
(Ap. 2), Montevideo.

NATIONAL

American Medical Association, Scientific Assembly, Section on Pediatrics

Chairman: Dr. John Aikman, 184 Alexander S
Rochester, N. Y.
Secretary: Dr. Gilbert J. Levy, 188 S. Bellevue Blvd
Memphis, Tenn.
In compliance with the request of the Office of Defen
Transportation and in the interest of the national w
effort a meeting will not be held in 1945.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

American Academy of Pediatrics

sident: Dr. Joseph S. Wall, 1864 Wyoming Ave. W., Washington, D. C.
retary: Dr. Clifford G. Grulee, 636 Church St., vanston, Ill.

American Hospital Association, Children's Hospital Section

irman: Dr. Joelle C. Hiebert, 299 Main St., Lewis-n, Maine.
retary: Dr. W. Franklin Wood, McLean Hospital, Waverly, Mass.

American Pediatric Society

sident: Dr. James L. Gamble, 300 Longwood Ave., oston.
retary-Treasurer: Dr. Hugh McCulloch, 325 N. uclid Ave., St. Louis.

Canadian Society for the Study of Diseases of Children

sident: Dr. R. R. Struthers, 906 Drummond Medical ldg., Montreal.
retary-Treasurer: Dr. Elizabeth Chant Robertson, ospital for Sick Children, Toronto.

Society for Pediatric Research

sident: Dr. Edward M. Bridge, 219 Bryant St., uffalo, N. Y.
retary: Dr. Mitchell I. Rubin, 1740 Bainbridge St., hiladelphia 46.

SECTIONAL

Intermountain Pediatric Society

sident: Dr. Eugene Smith, 385–24th St., Ogden, tah.
retary-Treasurer: Dr. W. C. Cheney, 837 Boston ldg., Salt Lake City.
ce: Salt Lake City General Hospital. Time: First hursday of each month, 8 p. m.

New England Pediatric Society

sident: Dr. Warren R. Sisson, 319 Longwood Ave., oston.
retary-Treasurer: Dr. James Marvin Baty, 1101 eacon St., Brookline, Mass.
ce: Boston Medical Library. Time: Four meetings year, occurring from September to May.

North Pacific Pediatric Society

sident: Dr. M. L. Bridgeman, 1020 S. W. Taylor St., Portland, Ore.
retary: Dr. C. G. Ashley, 833 S. W. 11th Ave., ortland, Ore.

Northwestern Pediatric Society

sident: Dr. Arild E. Hansen, University of Minne-sota, Minneapolis.
retary-Treasurer: Dr. Albert V. Stoesser, 205 W. University Hospital, Minneapolis.
ce: Minneapolis, St. Paul, Duluth and Rochester. Time: January, April, July and October.

Rocky Mountain Pediatric Society

sident: Dr. G. R. Fisher, 23 E. Pikes Peak Ave., Colorado Springs, Colo.
retary: Dr. Joseph H. Lyday, 1850 Gilpin St., Denver.

Southern Medical Association, Section of Pediatrics

Chairman: Dr. William Weston Jr., 1428 Lady St., Columbia, S. C.
Secretary: Dr. Angus McBryde, 604 W. Chapel Hill St., Durham, N. C.

STATE

Alabama Pediatric Society

President: Dr. Amas Gipson, 948 Forrest Ave., Gadsen.
Secretary-Treasurer: Dr. Ruth Berrey, 2021–6th Ave. N., Birmingham.

Arizona Pediatric Society

President: Dr. Vivian Tappan, San Clemente, Tucson.
Secretary: Dr. Hilda Kroeger, Arizona State Health Dept. (Maternal and Child Welfare Division), Phoenix.

Arkansas State Pediatric Association

Chairman: Dr. C. B. Billingsley, 1425 N. 11th St., Fort Smith.
Secretary: Dr. R. E. Weddington, 1425 N. 11th St., Fort Smith.

California State Medical Society, Section on Pediatrics

Chairman: Dr. William C. Deamer, University of California Hospital, San Francisco.
Secretary: Dr. Charles W. Leach, 2000 Van Ness Ave., San Francisco.

Florida State Pediatric Society

President: Dr. Ludo Von Meysenbug, Box 3356, Daytona Beach.
Secretary: Dr. Robert Blessing, 409 Blount Bldg., Ft. Lauderdale.
Place: Concurrent with state association meeting at time of convention.

Georgia Pediatric Society

President: Dr. T. F. Davenport, 104 Ponce de Leon Ave. N. E., Atlanta.
Secretary-Treasurer: Dr. Don F. Cathcart, 478 Peachtree St. N. E., Atlanta.

Hezekiah Beardsley Pediatric Club of Connecticut

President: Dr. Edward T. Wakeman, 129 Whitney Ave., New Haven.
Secretary: Dr. Herman Yannet, Southbury Training School, Southbury.
Time: Three meetings a year.

Illinois State Medical Society, Section on Pediatrics

Chairman: Dr. Craig D. Butler, 715 Lake St., Oak Park.
Secretary: Dr. A. J. Fletcher, 139 N. Vermilion, Danville.

Indiana State Pediatric Society

President: Dr. K. T. Knode, 1105 E. Jefferson Bldg., South Bend.
Secretary-Treasurer: Dr. Mathew Winters, 621 Hume Mansur Bldg., Indianapolis.
Time: Two meetings a year.

Iowa Pediatric Society

President: Dr. Mark L. Floyd, Children's Hospital, Iowa City.
Secretary-Treasurer: Dr. James Dunn, Davenport Bank Bldg., Davenport.

Medical Society of State of New York, Section on Pediatrics

Chairman: Dr. A. Clement Silverman, 608 E. Genesee St., Syracuse.
Secretary: Dr. Albert G. Davis, 307 Gas and Electric Bldg., Utica.

Medical Society of the State of Pennsylvania, Pediatric Section

Chairman: Dr. Elwood W. Stitzel, 403 Central Trust Bldg., Altoona, Pa.
Secretary: Dr. P. F. Lucchesi, Philadelphia Hospital, Philadelphia.

Michigan State Medical Society, Pediatric Section

Chairman: Dr. Charles F. McKhann, University Hospital, Ann Arbor.
Secretary: Dr. Mark F. Osterlin, Central Michigan Children's Clinic, Traverse City.

Mississippi State Pediatric Society

President: Dr. Harvey F. Garrison Jr., 315 E. Capitol Pl., Jackson.
Secretary: Dr. Guy Verner, 126 N. Congress St., Jackson.

Nebraska Pediatric Society

President: Dr. John L. Gedgoud, University of Nebraska Hospital, Omaha.
Secretary-Treasurer: Dr. Gerald C. O'Neil, 640 City National Bank Bldg., Omaha.
Place: As announced by committee. Time: Third Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

New Hampshire Pediatric Society

President: Dr. MacLean J. Gill, 14 N. State St., Concord.
Secretary-Treasurer: Dr. Ursula G. Sanders, 46 Pleasant St., Concord.
Time: Twice yearly.

North Carolina Pediatric Society

President: Dr. Arthur H. London, 1105 W. Main St., Durham.
Secretary: Dr. Jay M. Arena, 604 W. Chapel Hill St., Durham.

Oklahoma State Pediatric Society

President: Dr. Ben H. Nicholson, 301 N. W. 12th St., Oklahoma City.
Secretary: Dr. Luvern Hays, 108 W. 6th St., Tulsa.
Place: Oklahoma Club. Time: 6:30 p. m., fourth Friday of each alternate month from September to May, inclusive.

South Carolina Pediatric Society

President: Dr. Lonita Boggs, 301 E. Coffee St., Greenville.
Secretary-Treasurer: Dr. Hilla Sheriff, Wade Hampton Office Bldg., Columbia.

Texas Pediatric Society

President: Dr. F. H. Lancaster, 4409 Fannin St., Houston.
Secretary-Treasurer: Dr. John E. Ashby, 3610 Fairmount Ave., Dallas.

Virginia Pediatric Society

President: Dr. L. E. Sutton Jr., Medical College Hospital, Richmond.
Secretary: Dr. Emily Gardner, 1100 W. Franklin St., Richmond.

West Virginia State Medical Society, Section on Pediatrics

President: Dr. Andrew Amick, 1021 Quarrier St., Charleston.
Secretary: Dr. A. A. Shawkey, Professional Bldg., Charleston.

LOCAL

Academy of Medicine of Cleveland Pediatric Section

Chairman: Dr. J. D. Nourse, 10515 Carnegie Ave., Cleveland.
Secretary: Dr. I. B. Silber, 10465 Carnegie Ave., Cleveland.
Place: Cleveland Medical Library Bldg. Time: October, December, February and April.

Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. I. Nelles Silverthorne, 170 St. George St., Toronto, Canada.
Secretary: Dr. G. P. Hamblin, 2333 Bloor St. W., Toronto, Canada.

Bronx Pediatric Society

President: Dr. Jacob H. Turner, 121 W. 197th St., New York.
Secretary: Dr. Harry S. Pizer, 1726 Davidson Ave., New York.
Place: Concourse Plaza Hotel, 161st St. and Grand Concourse. Time: Second Wednesday of each month except June, July, August and September.

Brooklyn Academy of Pediatrics

President: Dr. Kenneth G. Jennings, 159 Midwood St., Brooklyn.
Secretary: Dr. Joseph Battaglia, 713 Bushwick Ave., Brooklyn.
Place: Granada Hotel. Time: Fourth Wednesday of each month except May, June, July, August and September.

Buffalo Pediatric Society

President: Dr. A. Wilmot Jacobsen, 187 Bryant St., Buffalo, N. Y.
Secretary: Dr. Richard A. Downey, 786 Forest Ave., Buffalo, N. Y.
Place: Children's Hospital, 219 Bryant St. Time: 8:30 p. m., first Monday of each month from September to June.

Central New York Pediatric Club

President: Dr. Edward J. Wynkoop, 501 James St., Syracuse.
Secretary Dr. Frank J. Williams, 58 S. Swan St., Albany.
Places: Various cities in New York. Time: Third Tuesday of April and September.

Chicago Pediatric Society

President: Dr. Morley D. McNeal, 2 N. Sheridan Rd., Highland Park, Ill.
Secretary: Dr. Henry G. Poncher, 1819 W. Polk St., Chicago.
Place: Children's Memorial Hospital, 710 Fullerton Ave. Time: Third Tuesday of each month, October to May, inclusive.

Cincinnati Pediatric Society

sident: Dr. Lloyd K. Felter, 3144 Jefferson Ave., incinnati.
retary: Dr. T. Selkirk, 3530 Reading Rd., Cincinnati.
ce: Children's Hospital, Elland Ave., Cincinnati.
ime: On call.

Dallas Pediatric Society

sident: Dr. Robert A. Knox, 3701 Fairmount Ave., allas 4, Texas.
retary-Treasurer: Dr. Gladys J. Fashena, 4585 Belrt Pl., Dallas 4, Texas.
ce: Children's Medical Center. Time: 1 p. m., second nd fourth Saturdays of each month.

Detroit Pediatric Society

sident: Dr. John J. Pollack, 650 Maccabees Bldg., etroit, Mich.
retary: Dr. Philip J. Howard, 2799 W. Grand Blvd., etroit, Mich.
ce: Wayne County Medical Society. Time: 8:30 m., first Wednesday of each month from October June, inclusive.

Fulton County Medical Society, Pediatrics Section (Atlanta, Ga.)

airman: Dr. Don F. Cathcart, 478 Peachtree St. N. E., Atlanta.
retary: Dr. Harry Lange, 478 Peachtree St. N. E., tlanta.
ce: Academy of Medicine, 38 Prescott St. N. E.
ime: Second Thursday of each month from October April, 8 p. m.

Houston Pediatric Society

sident: Dr. Raymond Cohen, 2300 Caroline St., Houston, Texas.
retary: Dr. Betty Moody, 526 Richmond Rd., Houston, Texas.
ce: College Inn, Houston. Time: Fourth Monday f each month.

Kansas City (Missouri) Pediatric Society

sident: Dr. Edwin H. Schorer, 1103 Grand Ave., Kansas City.
retary: Dr. H. E. Petersen, Kirkpatrick Bldg., St. oseph, Mo.
ce: Kansas City General Hospital. Time: On call.

Los Angeles County Medical Association, Pediatric Section

sident: Dr. Milo B. Brooks, 1033 Gayley Ave., West Los Angeles.
retary-Treasurer: Dr. Elena Boder, 1830½ Lucille Ave., Los Angeles 26.
ce: Los Angeles County Medical Headquarters, 1925 Wilshire Blvd. Time: Second Monday of February, April, June, October and December.

Medical Society of the County of Kings and the Academy of Medicine of Brooklyn, Pediatric Section

sident: Dr. Abraham M. Litvak, 1145 Eastern Parkway, Brooklyn.
retary: Dr. Harold Levy, 750 St. Marks Ave., Brooklyn.
ace: 1313 Bedford Ave., Brooklyn. Time: 9:00 p. m., ourth Monday of each month, October to April, nclusive.

Medical Society of the County of Queens, Inc., Section on Pediatrics

Chairman: Dr. Meyeron Coe, 217-02-91st Ave., Queens Village, N. Y.
Secretary-Treasurer: Dr. Edith A. Mittell, 144-38th Ave., Flushing, N. Y.
Place: Queens County Medical Bldg., Forest Hills, N. Y. Time: Third Monday of October, January, March and May.

Medical Society of the District of Columbia, Section on Pediatrics

President: Dr. Harry A. Spigel, 2647 Connecticut Ave., Washington, D. C.
Secretary-Treasurer: Dr. Perry W. Gard, 2520 Woodley Rd., Washington, D. C.
Place: Medical Society Bldg., 1718 M St. N. W. Time: 8 p. m., fourth Thursday of every month.

Memphis Pediatric Society

President: Dr. F. T. Mitchell, 376 S. Bellevue Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. Harry Jacobson, 1193 Madison Ave., Memphis, Tenn.
Place: John Gaston Hospital. Time: Quarterly.

Milwaukee Pediatric Society

President: Dr. M. G. Peterman, 411 E. Mason St., Milwaukee 2.
Secretary-Treasurer: Dr. B. J. Malnekoff, 3213 N. Marietta Ave., Milwaukee.
Place: Milwaukee Athletic Club. Time: Second Wednesday of each alternate month, beginning with February.

New York Academy of Medicine, Section of Pediatrics

Chairman: Dr. Howard Craig, 175 E. 79th St., New York.
Secretary: Dr. Alfred E. Fischer, 73 E. 90th St., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

Northern California Affiliates

President: Dr. Crawford Bost, 400 Post St., San Francisco.
Secretary: Dr. William A. Reilly, 384 Post St., San Francisco.
Time: Second Thursday of September, November, January, March and May.

Oklahoma City Pediatric Society

President: Dr. William M. Taylor, 1200 N. Walker St., Oklahoma City.
Secretary: Dr. G. R. Felts, 625 N. W. 10th St., Oklahoma City.
Place: Oklahoma Club. Time: Third Thursday of each month.

Philadelphia Pediatric Society

President: Dr. Carl Fischer, Greene and Coulter Sts., Germantown, Philadelphia.
Secretary: Dr. Sherman Little, 1740 Bainbridge St., Philadelphia.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday in January, March, May and November.

Pittsburgh Pediatric Society

President: Dr. John D. Sturgeon Jr., 22 N. Gallatin Ave., Uniontown, Pa.
Secretary-Treasurer: Dr. C. J. Stoecklein, Medical Arts Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate month from October to June, inclusive.

Richmond Pediatric Society

President: Dr. Stanley Meade, 913 Floyd Ave., Richmond, Va.
Secretary-Treasurer: Dr. Louise Galvin, 214 S. Boulevard, Richmond, Va.
Place: Richmond Academy of Medicine, 1200 E. Clay St. Time: 8 p. m., third Thursday of each month, except June, July and August.

Rochester Pediatric Society

President: Dr. Herbert Soule, 122 Rutgers St., Rochester, N. Y.
Secretary-Treasurer: Dr. Jerome Glaser, 300 S. Goodman St., Rochester, N. Y.
Place: Rochester Academy of Medicine or arrangement by program committee. Time: Third Friday of each month from October to May.

St. Louis Pediatric Society

President: Dr. Peter G. Danis, 634 N. Grand Ave., St. Louis 3.
Secretary-Treasurer: Dr. H. Ewing Wachter, 3720 Washington Ave., St. Louis.
Place: St. Louis Medical Society Bldg. Time: Third Thursday of each month from November to June.

Seattle Pediatric Society

President: Dr. Frederick B. Joy, Stimson Bldg., Se
Secretary: Dr. Sherod M. Billington, Medical D Bldg., Seattle.
Place: College Club. Time: Third Friday of month from September to June at 6:30 p. m.

Southwestern Pediatric Society

President: Dr. Jeanette Harrison, 1136 W. 6th Los Angeles.
Secretary: Dr. Henry F. Gallagher, 1930 Wil Blvd., Los Angeles.
Place: Jonathan Club of Los Angeles. Time: Wednesday in January, March, May, September November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Campbell Harvey, 35 W. Huron Pontiac, Mich.
Secretary: Dr. Harry A. Towsley, University of M igan, Department of Pediatrics and Communic Diseases, Ann Arbor, Mich.

Westchester County Medical Society, Pediatric Section (New York)

President: Dr. John B. Ahouse, 27 Ludlow St., Yonk N. Y.
Secretary-Treasurer: Dr. Elvira Ostlund, 64 High Rd., Rye, N. Y.
Place: Grasslands Hospital, Valhalla, N. Y. T Third Thursday in October, December, February April.

THE AMERICAN JOURNAL OF DISEASES OF CHILDREN is published by the American Medical Association as a medium for the advancement of knowledge of the diseases of children and for the publication of scientific investigation in this field.

Manuscripts for publication, books for review and correspondence relating to the editorial management should be sent to Dr. Clifford G. Grulee, Chief Editor, 636 Church Street, Evanston, Ill., or to any other member of the Editorial Board. Communications regarding subscriptions, reprints, etc., should be addressed, AMERICAN JOURNAL OF DISEASES OF CHILDREN, American Medical Association, 535 North Dearborn Street, Chicago 10.

Articles are accepted for publication on condition that they are contributed solely to the AMERICAN JOURNAL OF DISEASES OF CHILDREN. Manuscripts must be typewritten, preferably double spaced, and the original copy should be submitted. Zinc etchings and halftones will be supplied by the Association when the original illustrations warrant reproduction and when their number is not considered excessive.

Footnotes and bibliographies (the latter are used only in exhaustive reviews of the literature) should conform to the style of the *Quarterly Cumulative Index Medicus*. This requires, in the order given: name of author, title of article and name of periodical, with volume, page, month—day of month if the journal appears weekly—and year.

Matter appearing in the AMERICAN JOURNAL OF DISEASES OF CHILDREN is covered by copyright, but as a rule no objection will be made to its reproduction in a reputable medical journal if proper credit is given. However, the reproduction for commercial purposes of articles appearing in the AMERICAN JOURNAL OF DISEASES OF CHILDREN or in any of the other publications issued by the Association will not be permitted.

The AMERICAN JOURNAL OF DISEASES OF CHILDREN is published monthly. The annual subscription price (for two volumes) is as follows: domestic, $8.00; Canadian, $8.40; foreign, $9.50, including postage. Single copies are 85 cents, postpaid.

Checks, money orders and drafts should be made payable to the American Medical Association.

OTHER PERIODICAL PUBLICATIONS
of the American Medical Association

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION—Weekly. Covers all the medical sciences and matters of general medical interest. Illustrated. Annual subscription price (three volumes): domestic, $8.00; Canadian, $9.50; foreign, $12.00. Single copies, 25 cents.

ARCHIVES OF INTERNAL MEDICINE—Monthly. Devoted to the publication of advanced original clinical and laboratory investigations in internal medicine. Illustrated. Annual subscription price (two volumes): domestic, $5.00; Canadian, $5.40; foreign, $6.00. Single copies, 75 cents.

ARCHIVES OF NEUROLOGY AND PSYCHIATRY—Monthly. A medium for the presentation of original articles on nervous and mental diseases, with abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $8.00; Canadian, $8.40; foreign, $9.50. Single copies, 85 cents.

ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY—Monthly. Devoted to advancing the knowledge of and progress in cutaneous diseases and syphilis. Publishes original contributions and abstracts of the literature on these two subjects, transactions of the important dermatologic societies, book reviews, etc. Illustrated. Annual subscription price (two volumes): domestic, $8.00; Canadian, $8.40; foreign, $9.00. Single copies, 85 cents.

ARCHIVES OF SURGERY—Monthly. Devoted largely to the investigative and clinical phases of surgery, with monthly reviews on orthopedic and urologic surgery. Well illustrated. Annual subscription price (two volumes): domestic, $8.00; Canadian, $8.40; foreign, $9.00. Single copies, 85 cents, except special numbers.

ARCHIVES OF OTOLARYNGOLOGY—Monthly. A medium for the presentation of original articles on diseases of the ear, nose and throat, with abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 75 cents.

ARCHIVES OF PATHOLOGY—Monthly. A periodical devoted to the publication of original articles and general reviews in the field of pathology. Illustrated. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 75 cents, except special issues.

ARCHIVES OF OPHTHALMOLOGY—Monthly. Includes original articles on diseases of the eye, abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $8.00; Canadian, $8.40; foreign, $9.00. Single copies, 85 cents.

WAR MEDICINE—Monthly. Devoted to material on preparedness and scientific advancement in relation to military, naval, public health and allied services. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 75 cents.

QUARTERLY CUMULATIVE INDEX MEDICUS—Quarterly. A complete subject and author index to the worth while current medical literature of the world. Issued four times a year. Second and fourth volumes bound for permanent reference. Subscription price, calendar year: domestic, $12.00; Canadian, $14.00; foreign, $14.00.

AMERICAN MEDICAL ASSOCIATION

535 North Dearborn Street **CHICAGO 10**

CONTENTS

PAGE

Clinical Appraisal of Infants' Head Size. Julian D. Boyd, M.D., Iowa City 71

Immunity to Tetanus Induced by a Third Dose of Toxoid Four Years After Basic Immunization: Observations Based on a Study of Twenty-Five Allergic Children. M. Murray Peshkin, M.D., New York 83

Changes in the Vibratory Sense of Patients with Poliomyelitis As Measured by the Pallesthesiometer. William O. Frohring, B.S.; Paul M. Kohn, M.D.; James F. Bosma, M.D., and John A. Toomey, M.D., Cleveland 89

Normal Standards in the Treatment of Young Persons with Diabetes. Joseph H. Barach, M.D., Pittsburgh 92

Case Reports:

Erythroblastosis Fetalis in Mothers with Rh-Positive Blood: Report of Six Cases, with Comment on Isoimmunization with the "A" and "B" Agglutinogens. S. H. Polayes, Ph.B., M.D., Brooklyn 99

Hemorrhagic Uremia. M. G. Peterman, M.D., Milwaukee 103

Congenital Ileal Atresia with Gangrene, Perforation and Peritonitis in a Newborn Infant. Staged Operations: Obstructive Resection, Ileocolostomy and Excision of Exteriorized Ileum. Ernest E. Arnheim, M.D., New York 108

Correspondence:

Pygopagus Twins. M. A. Perlstein, M.D., Chicago 117

News and Comment 117

Abstracts from Current Literature 118

Book Reviews 133

Directory of Pediatric Societies 136